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Support Document for Proposed Rule on Friable Asbestos-Containing Materials in School Buildings. Health Effects and Magnitude of Exposure

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The Agency has determined that exposure to asbestos in school buildings poses a significant hazard to public health. Expousre to asbestos fibers can lead to serious and irreversible diseases. Friable asbestos-containing materials release asbestos fibers into the ambient environment. A sizeable proportion of schools contain asbestos-containing materials. In certain conditions these materials release fibers in concentrations which pose increased risks of developing the disease.

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Health Effects and Magnitude of Exposure

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Support Document
for Proposed Rule on
Friable Asbestos-Containing Materials in School Buildings

HEALTH EFFECTS AND MAGNITUDE OF EXPOSURE

October 1980

Office of Testing and Evaluation
Office of Pesticides and Toxic Substances
U.S. Environmental Protection Agency
Washington, DC 20460

1-A

When promulgating a rule concerning a chemical substance or mixture under the Toxic Substances Control Act (TSCA), the Administrator is required to publish a statement on the effects of that substance on health and the magnitude of exposure of human beings to that substance. This document is a preliminary statement of these findings in support of the rule "Friable Asbestos-Containing Materials in Schools Proposed Identification and Notification." It is a draft and is released for comment on its technical merit and policy implication.

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I. INTRODUCTION

Exposure to asbestos fibers can lead to numerous serious and irreversible diseases. Certain building materials in common use can release asbestos fibers into the atmosphere. In particular, friable asbestos-containing materials have been found to release fibers in concentrations which, if inhaled, are sufficient to increase the risk of developing such diseases. Some 3,000,000 students and 250,000 teachers and other staff regularly use public school buildings which contain friable asbestos-containing materials and which may contain such levels of contamination.

The Agency has determined that exposure to asbestos in school buildings poses a significant hazard to public health. This determination is based on the following considerations:

(1) the extent of use of friable asbestos-containing materials in schools,

(2) the number of diseases which epidemiologic studies have shown to be caused by exposure to asbestos,

(3) the evidence of elevated airborne concentrations of asbestos in schools and other buildings where friable asbestos-containing materials are present, and

(4) an estimate of the degree of risk posed by these elevated concentrations.

In addition, information on the identification of friable asbestos-containing materials and control measures that can be taken to reduce the release of and consequent exposure to asbestos fibers are presented.

II. USE AND PRESENCE OF FRIABLE ASBESTOS-CONTAINING MATERIALS IN SCHOOLS

Asbestos is a general term for a group of naturally occurring hydrated mineral silicates that separate into fibers. Asbestos minerals used commercially include: chrysotile, amosite, crocidolite, tremolite asbestos, actinolite asbestos, and anthophyllite asbestos.

Chrysotile is a serpentine mineral consisting of "layers" of SiO_4 linked by Mg ions. The other five minerals are amphiboles, which consist of "chains" of SiO_4 linked laterally by Ca, Mg, Fe, or Na ions.

Asbestos minerals have properties which make them common construction materials throughout the world. The construction industry used more than half of the 725,000 metric tons of asbestos consumed in the U.S. in 1976. Asbestos is incombustible, which makes it a good thermal and electrical insulator, and it has high tensile strength and moderate to good chemical resistance (Levine 1978). Its fibrous form adds cohesive strength to some materials. Asbestos fibers may be packed, woven, or sprayed.

Asbestos-containing materials can be friable; i.e., easily crumbled or pulverized. They may also be bound in a firm matrix such as cement or organic resins, however, and can be hard and resistant to damage.

A. Uses of Friable Asbestos-Containing Materials in Building Construction

Asbestos is used in buildings as a spray- or trowel-applied coating to building surfaces to retard fire, deaden sound, or decorate; it is also used in lagging on boilers and pipes, and in cement products, plasters, vinyl tile, and miscellaneous products such as lab table tops and ventilation hoods. Asbestos-containing sprayed- or trowelled-on materials were first used in the U.S. in 1935, when the material was found to be suitable for acoustical purposes and for decorative finishes in public buildings. In the 1950's, one of the most significant advances in the construction industry was the replacement of concrete with asbestos to protect structural steel against fire. Structural steel must be insulated to ensure that it does not become soft, bend, and collaspe during a fire. The replacement of concrete by asbestos greatly reduced the weight and bulk of large buildings (Sawyer 1979).

The amount of asbestos in the mixtures used in these applications varies widely. From 1% to 80% or more of asbestos, usually chrysotile or amosite or a mixture of the two, were combined with other fibers (including cellulose, mineral wool, or fiberglass), and cement or resinous binders. Table 1 shows the results of analyses of a variety of sprayed- or trowelled-on asbestos samples from schools in the U.S. (Battelle 1980). Nicholson et al. (1978a) reported similar concentrations for schools in New Jersey. (Paint present in these samples may have been applied at the time of spraying or during subsequent maintenance operations.)

Table 1. Site Sample Results from Battelie Bulk Sample
(Continued)

Sample No.	Location	Chrysotile (%)	Amosite (%)	Anthophyllite (%)	Other fibers	Nonfiber materials	Sample appearance
15-01A	Hall water damage		90		2% min. wool	5% calcite 3% opaque	Fibrous
01B	Hall water damage		80			10% gypsum, 5% glass, 5% opaque	
01C	Hall		50		30% min. wool	10% gypsum, 5% glass, 5% opaque	
01D	Hall		40		30% min. wool	20% gypsum 5% glass, 5% opaque	Fibrous
03A			20		20% min. wool	40% gypsum, 10% glass, 10% opaque	
03B		5	40		30% min. wool	20% calcite 5% glass, opaque	Fibrous
16-01B	Hall	5				50% opaque, 50% calcite, 40% opaque, 5% cotton	
02A	Hall wet damage	0			20% min. wool	20% calcite 60% opaque	Granular
02B	Hall wet damage	5				45% calcite, 50% opaque	
02C	Hall	10				50% calcite, 40% opaque	Granular
02D	Hall	10			50% fiber-glass	30% calcite, 50% opaque	Chunky-granular
03A	Music room	10			10% fiber-glass	25% calcite, 5% opaque, 5% cotton	Granular
03B	Music room	10			10% fiber-glass	40% calcite, 40% opaque	
04A	Cafeteria	5			10% fiber-glass	35% calcite, 10% opaque	Granular
04B	Cafeteria	5			10% fiber-glass	35% calcite, 60% opaque	
17-01A	Classroom	0				60% wood, 20% gypsum 10% opaque	Fibrous
01B	Classroom	0				60% wood, 20% gypsum 20% opaque	
02A	Art room	0				70% wood 20% gypsum, 10% opaque	Fibrous
02B	Art room	0				60% wood, 20% gypsum, 20% opaque, 50% wood	Fibrous

In the U.S., two principal methods have been used to apply formulations of mineral fibers, including asbestos, for building construction applications. In one method, dry fibrous material was pumped through a 2- to 4-in. hose. The hose conveyed the dry material to a nozzle at the actual site of application. As the dry material left the nozzle, it passed through the focus of a ring of fine water jets. The mixing took place at the focal point, approximately 4 to 8 in. from the end of the nozzle (Reitz 1972). The mixture was directed against the building surface from a distance of about 2 ft, and depths of application ranged up to 3 in. The material applied by this method often was fibrous in nature, rather than compact and granular. A coat of resin or paint frequently was incorporated to increase the cohesiveness of the final coating.

In the second process, the material was premixed with water in a hopper, and the resulting slurry was pumped to a nozzle and sprayed on the surface (Reitze et al. 1972). This usually resulted in a less fibrous, more compact material being applied. The depth of application generally did not exceed 1 in. (Barnes 1976).

Material that was trowelled-on had essentially the same composition as the sprayed-on materials, and it too was premixed with water. This material probably formed the densest, hardest coating of the three types. The depth of application usually did not exceed a fraction of an inch.

Asbestos also was applied to pipes and boilers in several ways. In some instances, a wet slurry similar to the above material was sprayed or trowelled on. In others, a blanket consisting principally of woven asbestos fibers was wrapped around the pipe and secured with plaster, tape, or a sprayed-on binder.

In 1973, EPA banned the use of spray-applied asbestos-containing material as insulation in buildings to prevent widespread contamination of the environment during spraying (EPA 1973). EPA amended this regulation in 1975 to include asbestos-containing pipe lagging, regardless of the method of application (EPA 1975). EPA extended this ban in 1978, ban to all uses of sprayed-on asbestos (EPA 1978). EPA also regulated the methods of removing asbestos from a building and disposing of the wastes generated by removal (EPA 1973). These regulations apply to "friable asbestos material," which is defined as material "that contains more than 1 percent asbestos by weight and that can be crumbled, pulverized, or reduced to powder, when dry, by hand pressure." For the purposes of regulating the spraying process, EPA defined "asbestos-containing" as containing >1% asbestos in bulk. Thus, the regulation does not preclude the use of materials contaminated by small amounts of asbestos ($\leq$1% in bulk) in spray formulations.

B. Presence of Friable Asbestos-Containing Materials in Schools

EPA has gathered information on the presence of friable asbestos-containing materials in public schools by surveying school districts and reviewing State, municipal and other programs to identify and control asbestos hazards.

Table 2 shows the results, as of April 1980, of an EPA survey of school districts regarding friable asbestos-containing materials. EPA mailed a guidance manual which included a survey form to school districts across the Nation in May, 1979. A copy of the survey form follows Table 2.

768 school districts containing 7,378 public schools (about 8% of the nation's total) responded to the survey. Of the 6,422 schools in these districts which were built or renovated between 1945 and 1973, 5,797 were inspected. 1,916, or 33% of the inspected schools that responded were identified as having friable asbestos-containing materials.

Although school districts across the country returned forms, districts in 7 States responded and less than 2% of the districts in 22 of the remaining States responded (Table 3).

EPA has preliminarily estimated that 8,545 public schools have friable asbestos-containing materials. These estimates are based on the survey responses and follow-up contacts with the reporting school districts, contacts with school districts that did not respond to the survey, and data supplied by New York City's program on asbestos in schools.

Table 2. U.S. Environmental Protection Agency, Office of Pesticides and Toxic Substances Asbestos Survey Report by School District

EPA Region	#SCH. IN DIST.	#SCH. BLT/REN 45–78	#SCH. INSP.	#SCH. USING PLM	#SCH. W/ ASB/ DATE	# SCH. EXPOSED PROB.	SQ. FT. EXPOSED ASBESTOS	#CHILD EXPOS.	#SQ. FT REMOVE/COST	#SQ. FT. ENCAP/COST	#SQ. FT. ENCLS/COST	#SQ. FT. DEFER/INSP.
I	45	32	42	10	10	7	9,500	501		1,200 2,250	5,000 19,000	4,500
II	69	62	72	10	4	8	7,073	560		1,000		6,073
III	3,307	3,180	2,666	357	1,574	267	2,414,320	102,113	671,953 12,512,089	721,527 6,508,988	100,435 7,155,785	1,172,854
IV	184	64	89	14	11	9	125,290	1,434	41,480 6,940,000			87,720
V	540	466	418	106	53	20	327,911	9,279	128,370 887,885	63,840 1,009,200	10 100	362,100
VI	828	675	588	70	33	15	356,562	8,402	51,190 1,876,600	269,317 25,353,650	125,000 125,000	106,291
VII	187	94	147	22	33	21	844,697	8,624		281,500 65,000	13,000	13,500
VIII	261	199	195	13	14	10	1,399,991	2,590	1,405 1,982,250	123,800 610,000	116,640 5,317,000	30,347
IX	755	670	612	53	79	36	286,677	5,721	112,920 543,566	15,200 1,400,400		27,300
X	1,202	980	968	94	105	43	528,970	58,923	143,157 576,566	142,248 866,400	5,244 2,875	386,968
National Totals	7,378	6,422	5,797	749	1,916	436	6,295,991	198,147	1,150,475 25,318,956	1,619,632 35,815,888	365,329 12,619,760	2,197,653

Note: See the following "Asbestos Survey Report", EPA Form 7710-29 (3-79), for full text of questions 4 through 12.

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EPA

U.S. ENVIRONMENTAL PROTECTION AGENCY

ASBESTOS SURVEY REPORT

(Survey of Activities to Control Asbestos–Containing Materials in School Buildings)

Form Approved
OMB No. 158–R–0165

GENERAL

This information is collected under the authority of the **Toxic Substances Control Act, Sections 6 and 8.** EPA is compiling information on the progress of State and local programs to control exposure to asbestos–containing materials in schools. This form should be used to periodically report information concerning the asbestos control activities in your school district. To obtain more forms, call this toll–free number: 800–424–9065 or in the Washington, D.C. area, call 554–1404. Data collected in this survey will be subject to the provisions of the Freedom of Information Act *(5 U.S.C. 552).*

MAILING INSTRUCTIONS

MAIL ONE COPY TO: The EPA Regional Asbestos Coordinator for your Region. *(For names and addresses see reverse side.)*

ALSO, please mail a copy to your official State asbestos program contact *(for name and address, call this toll–free number: 800–424–9065 or if in the Washington, D.C. area, call 554–1404).*

IDENTIFICATION

1. SCHOOL DISTRICT INFORMATION

NAME OF SCHOOL DISTRICT

CITY OR COUNTY

STATE

ZIP CODE

2. PERSON TO CONTACT REGARDING THIS REPORT

NAME *(last, first, & middle initial)*

OFFICIAL JOB TITLE

TELEPHONE NO. *(area code & number)* – –

DATE *(mo., day, & year)* – –

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SPECIFIC QUESTIONS

3. Has the school district submitted an EPA Asbestos Survey Report before?

☐ YES ☐ NO ☐ UNKNOWN

4. How many schools in the district were built or renovated between 1945 and 1978?

NUMBER OF SCHOOLS

5. As of ______ *(mo./yr.)*, how many schools in the district have been inspected for the presence of friable asbestos–containing materials?

NUMBER OF SCHOOLS

6. How many schools had bulk samples analyzed for asbestos with the EPA recommended technique of Polarized Light Microscopy?

NUMBER OF SCHOOLS

7. As of __________ *(mo./yr. of analysis)* for how many schools in the district was friable material analyzed as containing asbestos?

NUMBER OF SCHOOLS

8. (a) In how many schools was friable asbestos–containing material determined to present an exposure problem?
   (b) Approximately how many square feet of this material were found?
   (c) Estimate the number of children per school year exposed to this material. *(Multiply the percent of children exposed by the total number of enrolled students. e.g., An exposure problem in five classrooms may involve 15% of the total population of 700 students; 15% x 700 equals 105 students exposed.)*
   (d) Have the names of the children been recorded and retained for future reference?

a. NO. OF SCHOOLS

b. SQUARE FEET

c. NO. OF CHILDREN

d. NAMES RECORDED ☐ YES ☐ NO

Questions 9 through 11 refer to the friable asbestos–containing material that presents an exposure problem in Question 8.

9. (a) Approximately how many square feet of this material have been or will be removed?
   (b) What is the estimated total cost of removal?

a. SQUARE FEET

b. COST: $

10. (a) Approximately how many square feet of this material have been or will be encapsulated?
    (b) What is the estimated total cost of encapsulation?

a. SQUARE FEET

b. COST: $

11. (a) Approximately how many square feet of this material have been or will be enclosed?
    (b) What is the estimated total cost of enclosure?

a. SQUARE FEET

b. COST: $

12. (a) For approximately how many square feet of asbestos–containing material was action deferred?
    (b) Will this material be inspected periodically to determine if an exposure problem exists?

a. SQUARE FEET

b. PERIODIC INSPECTION ☐ YES ☐ NO

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13. What is the source of funding for the asbestos control activities in your district?

FUNDING SOURCE

14. When did *(or will)* the asbestos control activities in the district begin and end?

BEGINNING YEAR

ENDING YEAR

COMMENTS

EPA Form 7710–29 (3–79)

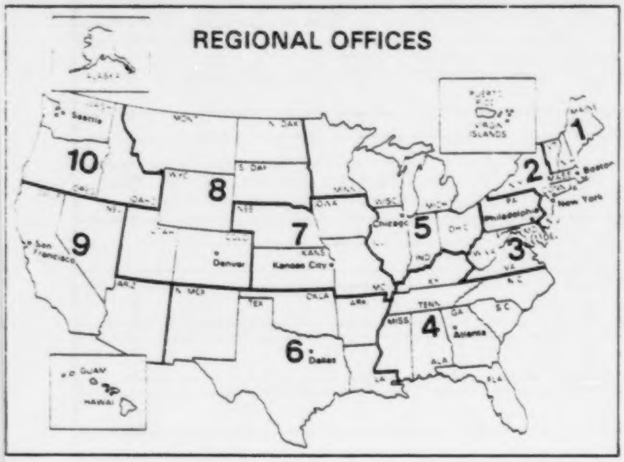
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Table 3. Respondents to EPA Survey for Asbestos-Containing Materials in Schools
(As of April 25, 1980)

State	No. of forms returned	Percentage of total districts that responded	State	No. of forms returned	Percentage of total districts that responded
Alaska	2	3.9	Nebraska	7	2.0
Alabama	0	0	Nevada	5	29.4
Arkansas	0	0	New Hampshire	1	0.2
Arizona	122	57.8	New Jersey	1	0.2
California	11	1.0	New Mexico	44	5.0
Colorado	3	1.7	New York	18	2.5
Connecticut	3	1.8	North Carolina	3	2.0
Delaware	8	50.0	North Dakota	24	7.8
Florida	1	1.5	Ohio	6	1.0
Georgia	1	0.5	Oklahoma	6	1.0
Hawaii	0	0	Oregon	14	25.9
Idaho	6	5.2	Pennsylvania	134	26.6
Illinois	5	0.5	Rhode Island	0	0
Indiana	0	0	South Carolina	1	1.1
Iowa	3	0.7	South Dakota	5	2.6
Kansas	3	0.9	Tennessee	2	1.3
Kentucky	1	0.5	Texas	22	10.0
Louisiana	4	6.0	Utah	4	10.0
Maine	1	0.7	Vermont	0	0
Maryland	0	0	Virginia	85	63.0
Massachusetts	2	0.8	Washington	119	39.3
Michigan	44	7.6	Washington, D.C. (citywide)	1	100.0
Minnesota	4	0.9	West Virginia	25	45.5
Mississippi	3	1.9	Wisconsin	2	0.5
Missouri	6	1.1	Wyoming	3	6.1
Montana	3	0.5			

The data for major school jurisdictions which reported inspection results for large portions of their schools compare favorably with the estimates. New York City reported that 180 of the 1,735 city schools inspected had sprayed-on friable asbestos-containing material in general use areas or 10.4% of the city schools. A 1978 statewide survey of 326 schools in Rhode Island revealed that 24 (8%) had sprayed-on asbestos material. In nine schools some degree of deterioration was noted (Faich 1980). Massachusetts' Special Commission on Asbestos in Schools and Public Buildings reported that walk-through surveys had been conducted in all 1,432 public schools in the State which were built or renovated between 1946 and 1972. 178 schools, 12%, were identified as containing "sprayed-on asbestos" (Commonwealth of Massachusetts 1978).

Several factors, in addition to the low response rate in some States, affect the validity of the estimates. First, the analysis is based on a small sample with a large response from one geographic area (EPA Region III). Second, this sample is not random, and it may reflect a bias due to the use of information from early respondents to the survey.

C. Number of Persons Exposed to Asbestos in Schools

Throughout the country, an estimated 3,000,000 students and 250,000 teachers, administrators, and other staff, including approximately 23,000 janitorial and maintenance workers are potentially exposed to airborne asbestos from friable asbestos-containing materials in public schools during the school year. An additional unknown number of persons may be exposed in private schools.

The number of exposed students was estimated from information contained in responses to the Agency's survey of school districts regarding friable asbestos-containing materials. The survey form included question 8(c): "Estimate the number of children per school year exposed to this (friable asbestos-containing) material." Respondents were instructed to consider whether only a portion of the school's population used the area in which friable asbestos-containing materials were found and to estimate the "exposed" population accordingly (see survey form).

Adjustments were made to this data base by contacting responding school districts, reviewing data from New York City, and contacting school districts which did not respond to determine whether the response to the survey was biased.

To complete the analysis, the sample school districts were clustered by metropolitan code (inner city, suburban, rural), EPA region, number of schools in the district, and number of students per district. Survey results were then extrapolated to the aggregate of public school districts to estimate the total number of students using areas likely to lead to exposure.

The number of exposed teachers was estimated on the basis of National Center for Education Statistics data that, nationwide, there is approximately 1 teacher per 20 students. Finally, the number of exposed janitorial and maintenance workers was estimated on the basis of the assumption that there are approximately two such staff persons for each of the 8,545 public schools with friable asbestos-containing materials.

D. Remaining Years of Use for School Buildings

School buildings are built to last for about 50 years, although during the late 1950's and the 1960's, slightly shorter lifetimes were expected. The 50-year estimate is a rule of thumb; no studies have been found that statistically or otherwise validate this approximation (Gardner 1980).

The schools most likely to have friable asbestos-containing materials were built between 1945 and 1973. Using the 50-year lifetime estimate, a school built in 1945 would have a remaining life of 15 years, one built in 1973 a life of 43 years. If the construction of the 8,545 schools with friable asbestos-containing materials was equally distributed among the years 1945 to 1973, the average expected remaining life would be 29 years. Factors that affect this estimated average are:

(1) spraying of asbestos was most popular in the late 1950's and in the 1960's;

(2) more schools were built during the 1950's and 1960's, to accommodate the postwar baby boom, than during 1945-1950;

(3) schools built in the 1950's and 1960's may not be expected to last as long as those built earlier or later;

(4) many schools are being closed across the nation because of declining enrollment.

The first two factors would increase the expected average remaining life of schools; the last two would reduce it. In view of the lack of definitive information on these factors as applied specifically to schools with friable asbestos-containing materials, an average remaining life of 30 years has been chosen.

III. ASSESSMENT OF RISK FROM ASBESTOS IN SCHOOLS

A. Introduction

Friable asbestos-containing materials that have been used in the construction of a large number of schools release asbestos fibers, and the Agency believes that occupants of these schools incur risks of developing diseases caused by exposure to such airborne fibers.

This section assesses the risks of adverse health effects and premature deaths from exposures to asbestos in schools. In making this assessment, it was necessary to identify the health hazards of asbestos exposure (Part B), to estimate the amount of asbestos to which occupants of schools are being or will be exposed, and to estimate the length of time and the number of occupants who are and will be exposed (Part C). This information, in turn, was used to estimate the number of people expected to die from asbestos-caused diseases as a result of exposure to "prevalent" (average) levels of asbestos in schools (Part D), if all asbestos materials currently in the schools remain in place until the buildings are no longer used.

The application of asbestos materials by spraying produces a friable coating. The fact that asbestos fibers may be released from these coatings was recognized as early as 1969 (Byrom, Hodgson, and Helms, 1969), which led to considerable concern that asbestos-caused diseases may develop in occupants of buildings containing the coatings (Reitze et al. 1972). Investigators found that fiber levels in these buildings varied widely because of a combination of many factors (Nicholson et al. 1978a, Sebastien et al. 1979a). The wide variation of asbestos concentrations in time and space means that no single measurement can determine prevalent levels of asbestos fibers (Nicholson et al. 1978a). Studies do show, however, that levels in buildings containing friable asbestos materials can frequently be very high ("peak" levels; see Table 13, Part C). Exposure to these levels and to lower, prevalent levels are predicted, as shown in Part D, to result in a considerable number of premature deaths among occupants of schools.

An evaluation of the risk requires combining estimates of asbestos concentrations in the buildings, the risk of disease due to a given exposure, the number of people exposed, and the duration of exposure. The accuracy of the risk evaluation is limited, however, because all of the available data are on a small number of areas sampled in a small number of buildings or on the risk of asbestos-induced disease in only a few populations. This limitation is dealt with in two ways: (1) in most cases, reasonable assumptions are made about how well the sampling data apply to possible situations in schools and the validity of these assumptions is discussed; (2) when reasonable assumptions cannot be made, cases are presented that give the lowest or highest reasonable estimate of risk. The accuracy of the risk estimates is defined in both of these ways.

Three sets of reasonable assumptions are made that give low, medium, and high estimates of the risk of mortality from exposure to the prevalent concentration of asbestos in schools. These estimates indicate that no fewer than 100 and no more than 7,000 premature deaths will be caused by exposure to prevalent levels of asbestos in schools if no controls are instituted. Additional premature deaths caused by short-term exposures to "peak" levels of asbestos also are very likely to occur. However, as explained in Part D, the number of these additional deaths as well as morbidity due to cancer and asbestosis cannot be estimated quantitatively.

B. Hazard Assessment

1. Introduction

The first step in assessing risk from asbestos in schools is to identify the adverse health effects arising from human exposure to asbestos. The evidence comes primarily from epidemiologic research. Persons exposed to asbestos were found in these studies to be at increased risk of developing specific diseases, thereby implicating the diseases as hazards of asbestos exposure. Indications of dose-response relationships in the studies support these findings.

The use of epidemiologic research to identify a disease as a hazard of asbestos exposure requires consideration of four major criteria: bias, confounding, chance, and biologic plausibility (Cole 1979). The proper design of studies and analysis of results to avoid misleading interpretation due to bias and confounding are explained in detail in many epidemiologic textbooks (e.g., MacMahon and Pugh 1970). The probability that apparent associations between asbestos exposure and specific diseases might be due to chance alone is distinguished by the application of standard statistical tests. In this assessment, it is considered biologically plausible that asbestos exposure can increase the risk of cancer at a given anatomic site if inhaled or ingested fibers can reach that site.

Epidemiologic studies may demonstrate dose-response relationships (increasing risk with increasing level of exposure) for asbestos-induced diseases in various degrees of detail. Many studies group exposure into only a small number of categories (e.g., "high," "medium," or "low"). These studies provide "qualitative" evidence of dose-response relationships and are briefly summarized. Other studies contain sufficiently detailed exposure data to examine in more detail the shape of dose-response curves within the range of observed exposures.

Part 2 below, identifies the following diseases on the basis of epidemiologic reasearch as hazards of asbestos exposure: lung cancer; pleural and peritoneal mesothelioma; cancers of the larynx, oral cavity, esophagus, stomach, colon, and kidney; and asbestosis.

The next step in the assessment is to identify factors that influence the degree of risk posed by asbestos exposure. In Part 3, smoking, age, and fiber type and size are discussed as possible factors that modify the degree of risk. As shown in Part B, the increase in lung cancer risk among smokers exposed to asbestos is greater than the sum of the separate increases produced by asbestos exposure alone and smoking alone. Smoking also may increase the risk of developing asbestosis. Because children have a greater remaining life span than adults, they may have a greater likelihood of developing asbestos-induced diseases. The overall influence of other age-related risk factors, however, is difficult to assess. All types of asbestos present in schools have been shown to be hazardous, and differences in fiber size and type are not likely to affect the risk assessment substantially.

2. Health Hazards of Asbestos Exposure

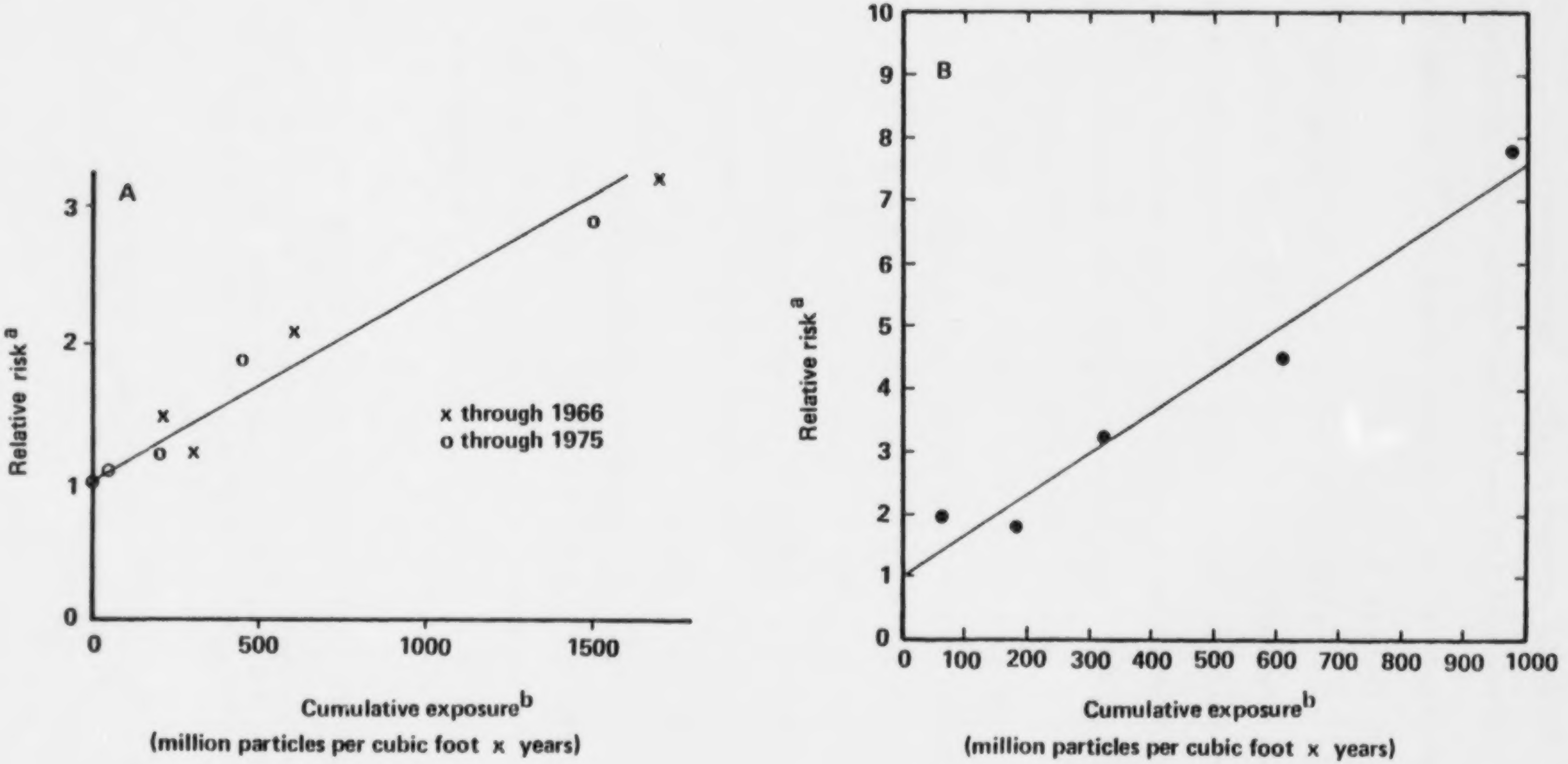
a. Lung Cancer

Epidemiologic studies demonstrate clearly that the risk of lung cancer is increased by exposure to asbestos (e.g., Doll 1955, Selikoff et al. 1964, Peto et al. 1977, Newhouse and Berry 1979). Several studies show qualitatively that the greater the exposure, the greater the increase in risk (Table 4). In addition, the authors of two studies of respiratory cancer mortality (predominantly due to cancers of the lung) among asbestos workers have drawn linear non-threshold dose-response curves to summarize their data (Figure 1).1/ According to this curve, all asbestos exposures, even those of very brief duration or very low intensities, intensities, increase risk of cancer.

1/ Figures 1 and 2 are presented simply to demonstrate the shape of the dose-response relationships in the two studies. These and similar dose-response curves appearing in this report should not be compared directly to each other, because substantial differences exist among study designs, measurement techniques, and exposure conditions. For instance, asbestos concentrations in Figures 1A and 1B were measured with the same type of instrument (midget impinger), but the method does not distinguish asbestos particles from other particles. Thus, the apparent difference in slope between the two curves could have resulted from a higher fraction of asbestos particles in samples taken in the asbestos products factory (Figure 1B) than in samples taken in the mining and milling facility (Figure 1B).

Table 4. Studies Showing Qualitative Dose-Response Relationships Between Asbestos Exposure and Increased Risk of Lung Cancer

Reference	Type of asbestos	Type of exposure	Measure of exposure
Newhouse and Berry (1979)	Mixed	Factory work	Intensity and duration
Hobbs et al. (1979)	Australian crocidolite	Mining and Milling	Duration
Meurman et al. (1979)	Anthophyllite	Mining and milling	Intensity
Wagoner et al. (1973)	Primarily chrysotile	Factory work	Duration
Mancuso and El-Attar (1967)	Primarily chrysotile	Factory work	Duration
Selikoff and Hammond (1975)	Amosite	Factory work	Duration
Nicholson et al. (1978b)	Primarily chrysotile	Factory work	Intensity
Hughes and Weill (1979)	Mixed	Factory work	Cumulative exposure



Source: Adapted from McDonald and Liddell (1979).

Source: Adapted from Henderson and Enterline (1979).

[a] Relative risk is the mortality rate in an exposed group divided by the rate in a comparison group. In Study A, the comparison group is the group of least exposed workers. In Study B, it is the general population.

[b] Units for cumulative exposure are not directly comparable among studies. See footnote on page 22.

Figure 1. Dose-response Curves for Respiratory Cancer Mortality in Two Groups of Asbestos Workers. A, Chrysotile miners and millers; B, retired asbestos production and maintenance workers.

The increases are directly proportional to cumulative exposure.[2/] This curve, and its use in predicting risk increases predicting risk increases at low exposure levels, is discussed in greater detail below in Part D.1.f.

Direct evidence of elevated lung cancer risk following low cumulative asbestos exposure is provided by a study of asbestos production workers (Seidman et al. 1979). In this study, men with less than 3 months of employment had a lung cancer mortality rate more than two times higher than that expected from general population rates. EPA has estimated that the average exposure level in the plant was 40,000,000 f/m^3 (EPA 1979a). Thus, an increase in lung cancer risk was detected epidemiologically following a cumulative exposure of less than 10,000,000 f-yr/m^3. Although it was achieved by relatively short-term exposure to high concentrations, this is the lowest level of cumulative asbestos exposure shown epidemiologically to lead to increased lung cancer risk.

b. Pleural and Peritoneal Mesothelioma

Malignant mesothelioma is an extremely rare type of cancer that appears as a thick, diffuse mass inside any of the serous membranes (mesothelia) that line body cavities.[3/] Considerable

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[2/] Cumulative exposure is calculated by multiplying the average concentration of asbestos in the air by the duration of exposure. When concentration is measured in fibers per cubic meter of air and duration is measured in years, the units for cumulative exposure are fiber-years/cubic meter (f-yr/m^3).

[3/] There is a benign form of mesothelioma (Taryle et al. 1976). This discussion concerns only the malignant form.

epidemiologic research (e.g., Wagner et al. 1960, Mancuso and Coulter 1963, Selikoff et al. 1965, Newhouse and Thompson 1965, Ashcroft and Heppleston 1970, Puntoni et al. 1976) has shown that exposure to asbestos can produce mesothelioma at two sites: the pleura, the serous membrane that surrounds the lungs and separates them from the thorax; and the peritoneum, the serous membrane that surrounds the abdominal organs and lines the abdominal and pelvic cavities.

Neither pleural nor peritoneal mesothelioma can be treated effectively, and both are nearly always fatal (Taryle et al. 1976, Kovarik 1976, Saijo et al. 1978). One-half of all patients die during the first year after diagnosis, and few patients survive longer than 2 years (e.g., Whitwell and Rawcliffe 1971, Rubino et al. 1972, Lumley 1976).

As in the case of lung cancer, a number of epidemiologic studies qualitatively demonstrate dose-response relationships between occupational asbestos exposure and the risk of mesothelioma (Table 5). In addition, Hobbs and colleagues (1979) found that the incidence of pleural mesothelioma among Australian crocidolite miners and millers increased in direct proportion to increasing duration of exposure. The linear trend and the occurrence of mesothelioma among the workers in this study who were exposed most briefly (<3 months) are reasonably compatible with a linear nonthreshold dose-response relationship (See Part D.1.f).

Table 5. Studies Showing Qualitative Dose-Response Relationships
Between Asbestos Exposure and Occurrence of Pleural and Peritoneal Mesothelioma

Reference	Type of asbestos	Anatomic site	Measure of exposure
McDonald et al. (1970)	Chrysotile	Pleura	Cumulative exposure
Newhouse and Berry (1979)	Mixed	Pleura/peritoneum combined	Duration and intensity
Hobbs et al. (1979)	Crocidolite	Pleura	Intensity
Selikoff (1977)	Amosite	Pleura/peritoneum separately	Duration

Pleural and peritoneal mesothelioma are considered "marker diseases" for asbestos exposure. A marker disease is one that is often, if not always, caused by a particular agent. In all cases of pleural and peritoneal mesothelioma, extremely rare types of cancer, there have been very strong suspicions that exposure to asbestos was the cause. In fact, as discussed below, close examination of individual case histories of mesothelioma patients usually provides evidence of some identifiable exposure to asbestos above ambient levels, even if only of brief duration or low intensity.

It is estimated that "apparently complete" case history information reveals some source of asbestos exposure above ambient levels for 85%-90% of all mesothelioma patients (Wagner et al. 1971). For some patients, however, "apparently complete" information is actually incomplete. Milne (1976) discovered that the last known occupations recorded on death certificates misleadingly indicated an absence of asbestos exposure for 66% of a series of mesothelioma patients later found, when their case histories were traced more diligently, to have been exposed. McEwen and colleagues (1971) found that the hospital records of 55% of another series of patients did not contain information on the asbestos exposures that these patients had, in fact, experienced. In addition, mesothelioma patients who, during personal interviews, were unable to recall experiencing any asbestos exposure were later found to have asbestos fibers in sections of their lung tissue taken at autopsy (Chen and Mottet 1978, Hourihane 1964). These studies strongly imply that significantly more than 90% of all persons with mesothelioma have been exposed to asbestos above the ambient outdoor exposure levels experienced by most urban dwellers (Selikoff and Lee 1978). Ambient exposure levels in urban and rural air may have been responsible for a substantial proportion of the <10% remaining cases. It is, therefore, reasonable to presume that all cases of mesothelioma in persons who have had previous asbestos exposure are the result of that exposure.

Given the status of pleural and peritoneal mesothelioma as marker diseases for asbestos exposure, the many well-documented cases that have followed extremely brief exposure to high concentrations of asbestos or long-term exposure to low concentrations provide evidence that risk is increased at these low levels of cumulative exposure. Table 6 lists a few of the cases of mesothelioma that have followed brief or low-intensity asbestos exposure both inside and outside the workplace. Table 7 lists 48 cases of mesothelioma that have occurred in persons sharing homes with asbestos workers. Table 8 lists 144 mesotheliomas that have occurred in persons who resided within a mile of an asbestos products factory, mine, or shipyard and who had no other known asbestos exposure. These case histories provide evidence that very brief or low-intensity exposure to asbestos can cause mesothelioma.

Table 6. Mesothelioma Occurring After Brief or Low-Intensity Asbestos Exposure

Reference	Anatomic site and nature of exposure
Lieben and Pistawka (1967)	1 pleural; helped replace plaster board during extensive remodeling of his house
	1 pleural; mixed and applied asbestos insulation to boilers in home for "a few hours"
	1 pleural; recycled asbestos filters in a brewery
	1 pleural; sawed pipe coverings at home
McDonald et al. (1970)	1 (site unspecified); handled asbestos sheet and pipe in a hardware store
Borow et al. (1973)	2 pleural; stock boys in asbestos products factory for 10 and 18 months, respectively
Newhouse (1973)	1 peritoneal; played on an asbestos factory waste pile as a child
Greenberg and Lloyd Davies (1974)	1 (site unspecified); relined and refitted clutches and brakes as hobby.
	1 (site unspecified); lived in a house largely composed of asbestos sheeting
	1 (site unspecified); worked on and lived adjacent to a chicken farm with asbestos-cement buildings
	1 (site unspecified)); sawed asbestos-cement sheets for 1 day to construct two sheds
Nurminen (1975)	1 pleural; did repair work on own house and handled asbestos boards
Jones et al. (1976)	1 (site unspecified); inspector at a gas mask assembly plant, did not handle asbestos pads used in assembly of masks
Arul and Holt (1977)	1 pleural; resided near an asbestos products factory for 2 years
Bruckman et al. (1977)	1 (site unspecified); toll collector
Whitwell et al. (1977)	2 pleural; filled gas mask cannisters with crocidolite for 6 months
Cochrane and Webster (1978)	1 pleural; jeweller, occasionally cut sections from a roll of asbestos textile
Seidman et al. (1979)	1 pleural and 1 peritoneal; worked in an amosite products factory for less than 9 months

Table 7. Mesothelioma Occurring in Persons
Sharing Households with Asbestos Workers

Reference	No. of mesotheliomas
Anderson et al. (1976)	37[a]
Vianna and Polan (1978)	7
Hobbs et al. (1979)	2
Edge and Choudhury (1978)	1
Hain et al. (1974)	1
Total	48

[a] Total includes cases reviewed from reports other than those listed.

Table 8. Mesothelioma Occurring in Persons Residing Near Point Sources of Asbestos Emissions

Reference	No. of mesotheliomas
Hain et al. (1974)	105[a]
Cochrane and Webster (1978)	13
Wagner et al. (1960)	13
Borow et al. (1973)	2
Greenberg and Lloyd Davies (1974)	10
Arul and Holt (1977)	1
Total	144

[a] Total includes cases reviewed from reports other than those listed.

c. Other Cancers

The scientific evidence reported below supports the identification of cancers of the larynx, oral cavity, esophagus, stomach, colon, and kidney as hazards of asbestos exposure.

Three cohort studies of asbestos workers (Newhouse and Berry 1979, Selikoff et al. 1979a, Rubino et al. 1979) and two case-control studies (Stell and McGill 1973, Morgan and Shettigara 1976) found increases in the risk of larynx cancer following exposure to asbestos. In one of the studies (Rubino et al. 1979), the risk increased with increasing cumulative asbestos exposure, an indication of a possible dose-response relationship.

The rates of mortality due to cancers of the esophagus and oral cavity the latter comprised of the (buccal cavity and pharynx) were elevated in a group of 17,800 asbestos insulation workers, compared with the rates in a group of other blue-collar workers (Hammond et al. 1979, Selikoff et al. 1979a). These cancers, like cancer of the larynx, have been shown to be related to cigarette smoking (Hammond 1966). To allow for this association, Hammond and colleagues (1979) accounted for the smoking habits of the insulation workers and the comparison group.

The asbestos insulation workers had higher stomach cancer mortality rates than the comparison group (Hammond et al. 1979). In addition, stomach cancer rates were elevated in a group of amosite production workers (Selikoff and Hammond 1975). In the latter group, the risk of stomach cancer increased with duration of asbestos exposure, an indication of a possible dose-response relationship.

A clear excess risk of colon cancer was reported in a group of 632 asbestos insulation workers in New York and New Jersey (Selikoff 1977a). Mortality rates for cancers of the colon and rectum combined were significantly elevated among the larger group of 17,800 asbestos insulation workers (Hammond et al. 1979) and among the amosite factory workers (Selikoff and Hammond 1975). Because the results for rectal cancer were not reported separately in the latter two studies, only cancer of the colon can be said to be a hazard of asbestos exposure at this time.

The large group of asbestos insulation workers also experienced an increase in kidney cancer mortality (Hammond et al. 1979). This epidemiologic finding and the corroboration lent by an experiment in which an excess of kidney cancer was seen in rats fed ground, paper-based beverage filters containing 53% chrysotile asbestos (Gibel et. al 1976) lead to the conclusion that kidney cancer should be considered a hazard of asbestos exposure.

Inhaled asbestos can be expected to reach each of the anatomic sites at which increased risks of cancer were shown in the epidemiologic studies discussed above. The process of respiratory clearance results in exposure of the larynx, oral cavity, esophagus, stomach and colon to inhaled asbestos fibers.[4/] Exposure of the kidneys was shown directly when chrysotile asbestos was found in the kidneys of rats (Cunningham et al. 1977) and a baboon (Hallenbeck and Patel-Mandlik 1978) that had been fed the fibers. Indirect evidence that the kidneys become exposed was provided by the detection of fibers in the urine of persons who drank water contaminated with the fibers (Cook and Olson 1979).

d. Asbestosis

Exposure to airborne asbestos produces a chronic noncancerous disease of the lungs that, in its severest form is called asbestosis. As implied by its name, the disease is caused solely by exposure to asbestos. It is characterized by a hardening and thickening of lung tissue that is called fibrosis. The rigidity produced by this process restricts the normal movement of the lungs. Asbestosis is irreversible and there is no effective treatment (Becklake 1976, Selikoff and Lee 1978). In advanced stages, the disease can be fatal. In a study of mortality among asbestos textile workers employed under extremely dusty conditions (Doll 1955), 63% of the death certificates listed noncancerous respiratory disease in conjunction with asbestosis as the cause of death.

[4/] The airways of the lungs are lined with a layer of mucus that is moved along by cilia, hairlike structures attached to the free surface of the cells on the airway surfaces. Inhaled particles that become embedded in the mucus eventually are cleared to the oral cavity, where they are swallowed or expectorated.

Asbestosis is a progressive disease that has various degrees of severity (Berry and Lewinsohn 1979). Asbestos exposure can continue to cause damage to the lungs even after direct exposure has ceased (Becklake et al. 1979). Symptoms likely to prompt an exposed individual to seek medical care, such as loss of breath or a bluish discoloration of the skin,[5/] do not appear until well after severe oxygen deprivation has occurred (Harries 1973, Robin 1979). In order to detect progressing asbestosis (i.e., the less severe stages of the disease), exposed individuals must be examined for clinical and diagnostic signs.

Most often, medical examinations of persons exposed to asbestos include chest x-rays and a physical examination that includes a determination of the presence or absence of crepitations, the abnormal lung sounds that are characteristic of asbestosis (Leathart 1968, Forgacs 1967, 1969). Unlike lung function tests, which are conducted less frequently, crepitations and abnormal x-ray findings do not indicate directly that health is impaired. Instead, they show that the disease process has begun. For example, persons with crepitations have a high probability of suffering later decrements in lung function (Berry et al. 1979). Often, persons with lung damage visible on x-rays already have impaired lung function (Jodoin et al. 1971, Selikoff and Lee 1978). Abnormal x-ray findings also indicate that a person is at high risk of subsequently developing more severe

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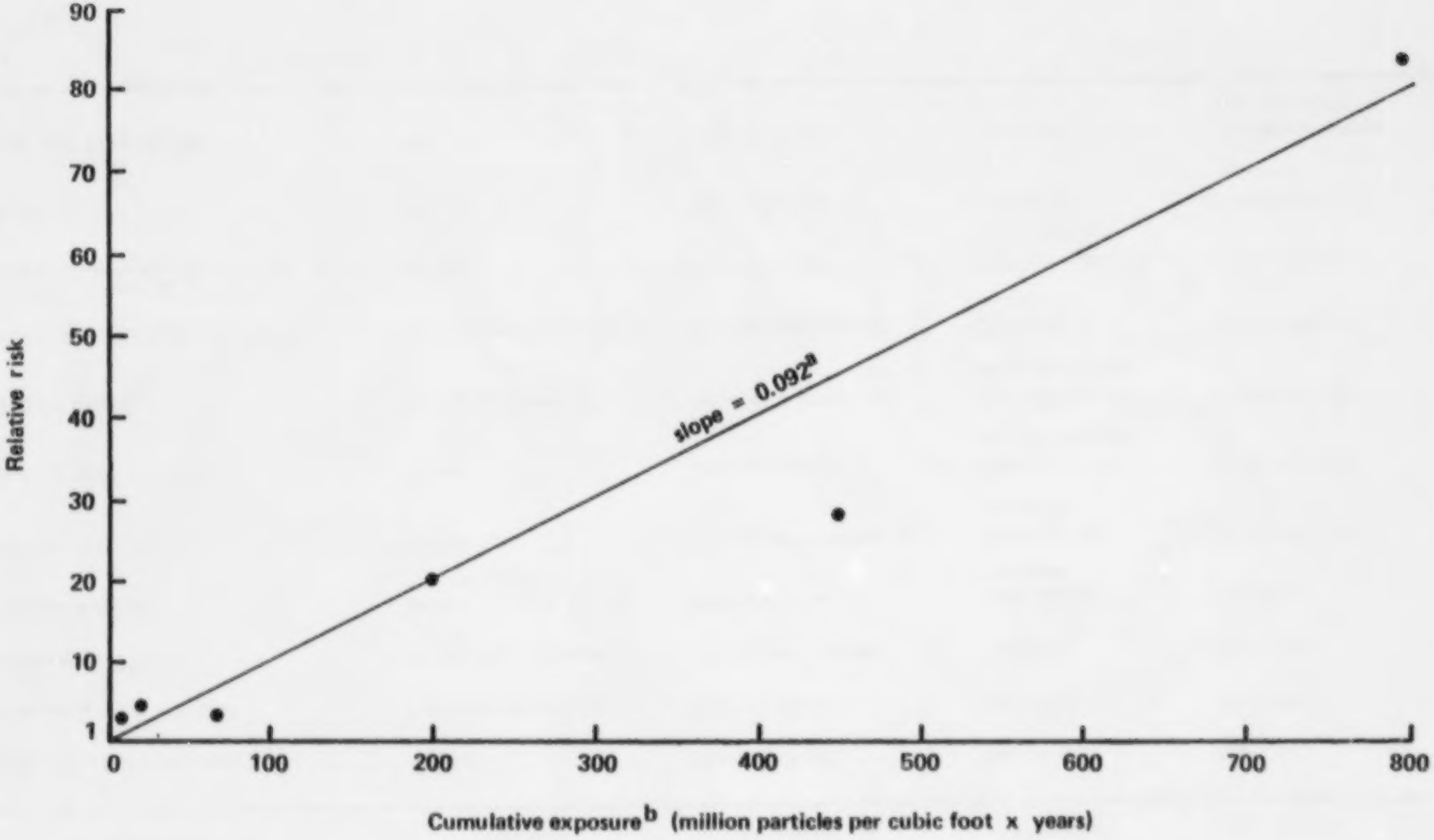
[5/] This discoloration (called cyanosis) is due to an excessive concentration of nonoxygenated hemoglobin in the blood.

stages of asbestosis. For instance, Liddell (1979) found that asbestos miners and millers with lung damage detectable on x-rays later experienced an asbestosis mortality rate nine times greater than that experienced by workers with normal x-rays. Consequently, although some researchers reserve the term "asbestosis" for advanced stages of the disease [e.g., "clinical" asbestosis (Murphy et al. 1971) or "certified" asbestosis (McVittie 1965)], crepitations, x-rays findings of lung damage, and measurements indicating decreased lung function are each considered signs of asbestosis in the following discussion.

A large number of occupational studies have used the various measures of asbestosis to demonstrate dose-response relationships. The studies in Table 9 show qualitatively that the risk of asbestosis rises with increasing asbestos exposure. McDonald and colleagues (1979) described the dose-response curve for asbestosis mortality among Canadian chrysotile miners and millers as a linear relationship (Figure 2), although they cautioned against extrapolation to very low exposure levels. An earlier study of x-ray-detectable lung damage among South African crocidolite miners and millers (Sluis-Cremer and duToit 1973) is consistent with this finding (Figure 3), as is a very recent study of asbestos textile workers in the United Kingdom (Berry and Lewinsohn 1979). Data from the latter study are used in Figure 4 to draw dose-response curves for three stages of asbestosis (crepitations, "possible" asbestosis, and "certified" asbestosis).

Table 9. Studies Showing Qualitative Dose-Response Relationships Between Asbestos Exposure and Various Measures of Asbestosis

Reference	Type of asbestos	Type of exposure	Measure of exposure	Measure of asbestosis
Selikoff and Hammond (1975)	Amosite	Factory Work	Duration	Mortality
Nicholson et al. (1979b)	Primarily chrysotile	Factory Work	Intensity	Mortality
Hobbs et al. (1979)	Australian crocidolite	Mining and milling	Duration	Incidence
Lacquet (1979)	Mixed	Factory work	Cumulative exposure	Incidence
Selikoff (1977b)	Chrysotile	Mining and milling	Duration and intensity	X-ray changes
Selikoff et al. (1979b)	Mixed	Shipyard work	Duration from exposure onset	X-ray changes
Selikoff (1977c)	Primarily chrysotile	Insulation work	Duration from exposure onset	X-ray changes
Sluis-Cremer and duToit (1973)	Amosite and crocidolite	Mining and milling	Cumulative exposure	X-ray changes
Baselga-Monte and Segarra (1978)	Mixed	Factory work	Mean cumulative exposure	X-ray changes
Harf et al. (1979)	Mixed	Spray application	Duration	Decreased vital capacity
Ayer and Burg (1978)	Mixed	Factory work	Duration	Decreased forced vital capacity

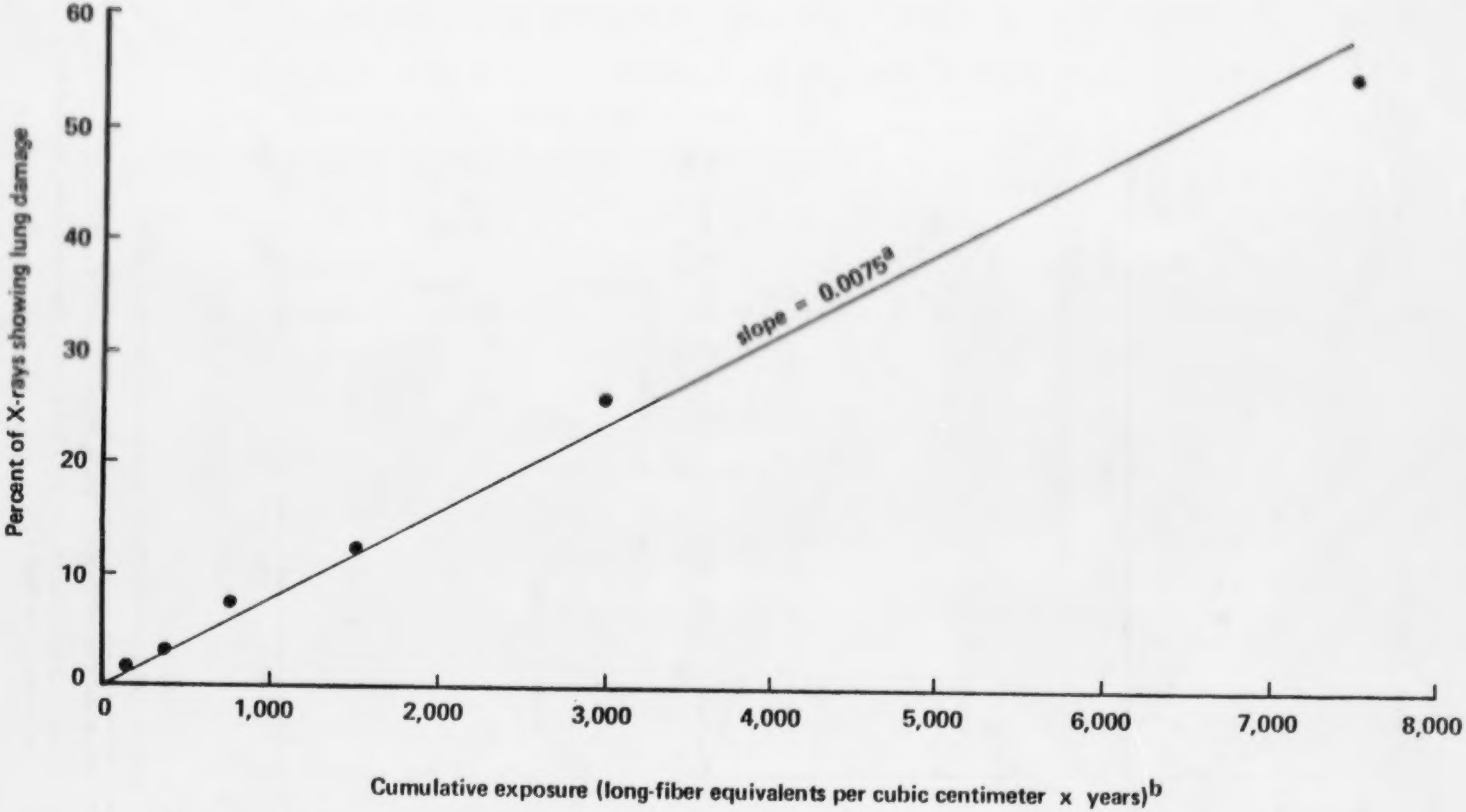


Source: McDonald, as reported Acheson and Gardner (1979).

[a] Slope determined by the formula, slope $=\Sigma x(y-1)/\Sigma x^2$.

[b] Units for cumulative exposure are not directly comparable among studies. See footnote on page 22.

Figure 2. Dose-response Curve for Asbestosis Mortality in a Group of Chrysotile Miners and Millers.

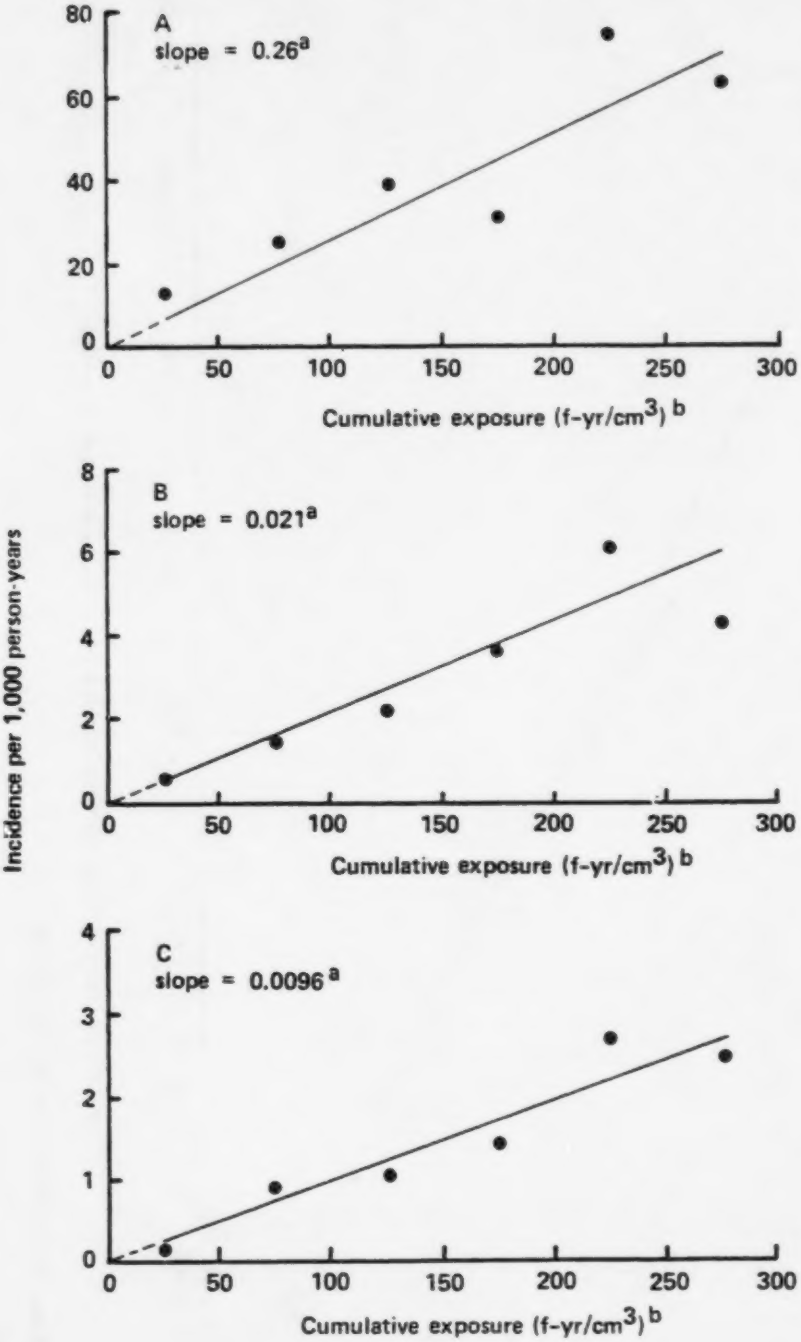


Source: Sluis-Cremer and duToit (1973).

[a] Slope determined by the formula, slope = $\Sigma xy/\Sigma x^2$.

[b] Converted by the authors from concentrations measured in million particles per cubic foot x years. Units for cumulative exposure are not directly comparable among studies. See footnote on page 22.

Figure 3. Dose-response Curve for X-ray Signs of Asbestos in a Group of South African Miners and Millers of Amosite and Crocidolite.



Source: Berry, as reported in Acheson and Gardner (1979).

[a] Slopes determined by the formula, slope = $\sum xy / \sum x^2$.

[b] Units for cumulative exposure are not directly comparable among studies See footnote on page 22.

Figure 4. Dose-response Curves for (A) Crepitations, (B) Possible Asbestosis and (C) Certified Asbestosis in a Group of Asbestos Textile Workers

Possible asbestosis represents a judgment by the factory medical officer, based on periodic physical examinations and x-rays, that the disease has progressed to the extent that a worker should move to a less dusty job. The diagnosis of certified asbestosis qualifies a patient for workmen's compensation (McVittie 1965). Without extrapolation, the curves in Figure 4 show the following annual incidence rates of asbestosis for workers with previous cumulative exposure of approximately
25 f-yr/cm^3:

certified asbestosis	2 cases/10,000 workers/year
possible asbestosis	5 cases/10,000 workers/year
crepitations	65 cases/10,000 workers/year.

This represents the lowest level of cumulative asbestos exposure at which severe forms of asbestosis have been detected.

These studies of dose-response relationships imply that the risk of asbestosis is proportional to cumulative asbestos exposure. Because the curves do not demonstrate a "no-adverse-effect level" of exposure, signs of asbestosis may well result from exposure levels lower than those present in the asbestos factories, mines, and mills that were studied.

The above implication is borne out by the results of studies that show that signs of less severe stages of asbestosis can occur in individuals exposed to asbestos outside the workplace. The most important results are those reported by Anderson and co-workers (1979), who found a high prevalence of lung abnormalities on the x-rays of children and other persons living in the same households as asbestos workers (Table 10). Persons sharing

Table 10. Lung Abnormalities Detected on X-rays of Persons Sharing Households with Asbestos Workers

Group	No. of persons examined	No. of x-rays with one or more abnormality
Controls	325	15 (4.6%)
All household contacts	679	239 (35.2%)[a]
Sons and daughters only	375	109 (29.1%)[a]
<1 year of exposure only	192	47 (24.5%)[a]

Source: Anderson et al. (1979)

[a] Probability that the difference from control value resulted by chance alone is less than 0.001 (Two-tailed chi square test. See Fleiss 1973).

households for less than 1 year with persons actively employed as asbestos workers had a percentage of abnormalities five times higher than that of controls. The asbestos concentrations in these homes are not known, but they are presumed to have been many times lower than those to which the workers were exposed at their places of employment. Thus, persons sharing households for less than 1 year with persons actively employed as asbestos workers had very low levels of cumulative exposure.

Another recently reported study concerns office workers whose only known asbestos exposure was from sprayed-on insulation materials in office buildings in Paris (Awad et al. 1979). These individuals received medical examinations that included a determination of the presence of "crackling rales" (crepitations). Of office workers employed for 10 or more years in building areas with "low protection" but no "specific exposure," only 0.4% had crepitations. The prevalence was three times higher (1.3%) among workers who were present during construction of the building but who, at the time of the survey, worked in buildings free of asbestos contamination. The highest prevalence (2.5%) was found among employees having direct contact with "ceilings, sheaths, cupboards, etc." coated with asbestos-containing materials. These results are no reported completely and, because of the small number of persons with crepitations, cannot be ruled out the possibility that these findings should be attributed to chance. Nevertheless, if validated, they will form the first direct evidence of asbestosis among occupants of buildings that, like many school buildings in the United States, were constructed with sprayed-on asbestos-containing materials.

In South Africa, Sluis-Cremer and duToit (1979) found lung abnormalities characteristic of asbestos exposure (e.g., pleural calcification) on the x-rays of nonworkers residing near asbestos mines. The prevalence increased with duration of residence in the area, another demonstration of a dose-response relationship.

The three studies of nonoccupational exposure cited above support the inference from occupational dose-response curves that lung damage characteristic of asbestosis can occur in persons exposed to asbestos concentrations lower than those in occupational settings. The two best sources of information for predicting whether signs of asbestosis can result from asbestos exposure levels found in schools would be the findings among household contacts of asbestos workers (Table 10) and the dose-response curves in Figure 4. Unfortunately, the absence of data on asbestos concentrations in workers' homes prevents a direct comparison with the situation in schools. The lowest level of cumulative exposure actually measured in Figure 4 (25 f-yr/cm^3) is approximately 100 times the highest estimate for adults employed in school buildings (using a conversion factor of 1 f/cm^3 = 33,000 ng/m^3; see Table 18). [Unlike the assessment of cancer risks, in which extrapolation is warranted by the current scientific understanding of carcinogenic processes and by regulatory policy, an extrapolation of asbestosis risks over two orders of magnitude of cumulative exposure may be unduly speculative.] Some noncancerous lung damage probably will result from asbestos exposure in schools, but the extent of damage cannot be predicted with a reasonable degree of confidence.

e. Summary and conclusions

Epidemiologic research has identified cancers of the lung, pleura, peritoneum, larynx, oral cavity, esophagus, stomach, colon and kidney as hazards of asbestos exposure. Inhalation of asbestos also produces the non-cancerous lung disease asbestosis. Dose-response relationships (increasing risk correlated with increasing asbestos exposure) have been shown or suggested for cancer of the lung, larynx, and stomach, pleural and peritoneal mesothelioma, and asbestosis. Two studies of respiratory cancer among asbestos workers provide results compatible with linear nonthreshold dose-response curves.

These dose-response studies imply that asbestos exposure can increase the risk of cancer at lower exposure levels than those studied. This expectation is supported by evidence of adverse health effects resulting from relatively low levels of asbestos exposure. Increased lung cancer risk has been observed among workers exposed to asbestos for the equivalent of 5 years at the current workplace standard of 2,000,000 f/m^3. Mesothelioma, a "marker disease" for asbestos exposure, has occurred in persons with exposures as brief as 1 or 2 days and in persons with steady exposures as low as those found in the homes of asbestos workers and in neighborhoods around asbestos mines, products factories, and shipyards. X-ray signs of asbestosis have been detected among persons sharing households with actively employed asbestos workers for less than a year. Linear nonthreshold dose-response curves predict that asbestos exposure in schools will produce adverse health effects (see Part D). This prediction is consistent with the occupational dose-response curves and with the observation of increased risks of disease at exposure levels lower than those found in the workplace.

Part B 3, below, identifies factors that influence the degree of increased risk posed by asbestos exposure. Part D, estimates risks of cancer mortality expected to result from exposure to asbestos in school buildings.

3. Factors that Modify the Risk of Asbestos-Induced Disease

a. Smoking

The major factor affecting the risk of asbestos-induced lung cancer, other than the intensity and duration of asbestos exposure, is the smoking habits of exposed individuals. Although, as shown below, asbestos exposure alone and cigarette smoking alone can each cause lung cancer in humans, the combined effects of cigarette smoking and asbestos exposure produce an increase in lung cancer risk that is greater than the sum of the increases produced by the two agents independently. In one study, a group of 283 asbestos insulation workers who smoked had a lung cancer mortality rate approximately 90 times greater than the rate they would have had if they had been neither smokers nor asbestos workers (Selikoff et al. 1968). In a more recent study of 17,800 asbestos insulation workers, the rate was 50-60 times greater (Hammond et al. 1979). As discussed below, this latter study also showed that the combined effect of smoking and asbestos exposure exceeded the sum of their separate effects, an indication that the effects of asbestos and smoking interact or modify one another in some way.

In the earlier study of asbestos insulation workers (Selikoff et al. 1968), there were no lung cancer deaths among 87 nonsmokers. This study prompted speculation that asbestos might increase lung cancer risk only in smokers (Cole and Goldman 1975, Hoffmann and Wynder 1976). The current evidence, however, shows that asbestos exposure induces lung cancer in smokers and nonsmokers alike. The recent study by Hammond and colleagues (1979), found a fivefold increase in the risk of lung cancer among 891 nonsmoking asbestos insulation workers. Because it covered a larger group of nonsmoking workers over a longer follow-up period, the study of Hammond et al. had a higher probability of detecting an increase in risk than the earlier study. Asbestos exposure, therefore, increases lung cancer risk even in the absence of cigarette smoking (Selikoff and Hammond 1979).

In the study by Hammond et al., the asbestos workers who smoked cigarettes could have avoided about the same increase in lung cancer risk if they had not been asbestos workers as they could have if they had not been smokers. In 1978, Selikoff supplied EPA with a set of unpublished data from this study that enables estimates to be made of the proportion of lung cancer deaths among the cigarette-smoking asbestos workers that can be attributed to smoking alone, asbestos alone, interaction of the effects of asbestos and smoking, and unknown factors (Table 11). Estimates were made of the number of deaths that could be expected among the smoking asbestos workers if they had been neither smokers nor asbestos workers (E_1), if they had smoked but

Table 11. Observed and Expected Lung Cancer Deaths ≥ 20 Years from Onset of Exposure in a Group of Asbestos Workers with a History of Cigarette Smoking.

Lung cancer deaths	No. of deaths[a]
Observed (0)	305
Expected on the basis of:	
Nonsmoking non-asbestos workers (E_1)	4.4
Smoking non-asbestos workers (E_2)	57.5
Nonsmoking asbestos workers (E_3)	35.0
Attributable to:	
Factors other than smoking or asbestos (E_1)	4.4 (1.4%)
Smoking alone ($E_2 - E_1$)	53.1 (17.4%)
Asbestos alone ($E_3 - E_1$)	30.6 (10.0%)
Asbestos /smoking interaction $(0 - [E_1 + (E_2 - E_1) + (E_3 - E_1)])$	216.9 (71.1%)

Source: Unpublished data supplied to EPA by Selikoff (1978)

[a] The most recently published results from this study (Selikoff et al. 1979a, Hammond et al. 1979) report $0 = 306$, $E_1 = 4.7$; E_2 and E_3 were not reported. We are requesting updated figures for 0, E_1, E_2, and E_3 from these researchers.

had not been exposed to asbestos (E_2), and if they had never smoked but had been exposed to asbestos (E_3). For E_1, the lung cancer mortality rates of a group of nonsmoking non-asbestos workers from a large study (Hammond 1966) sponsored by the American Cancer Society (ACS) were used. For E_2, the rates for smokers in the ACS study were used. The rates of the nonsmoking colleagues of the smoking asbestos insulaton workers were used to derive E_3.

The results, summarized in Figure 5, are nearly identical to the estimates derived by Lloyd (1979) using published data from the same study and a different method of derivation. Less than 2% of the lung cancer deaths among the cigarette-smoking asbestos workers were attributable to causes other than smoking and asbestos exposure; over 70% were the result of some sort of interaction between the effects of the two agents. The most important implication is that approximately 81% of the lung cancer deaths could have been prevented if none of the men had been asbestos workers and about 88% could have been prevented if none had been smokers. Thus, from a preventive standpoint, the impacts of smoking and asbestos exposure on lung cancer risk were approximately equal in this group of workers.

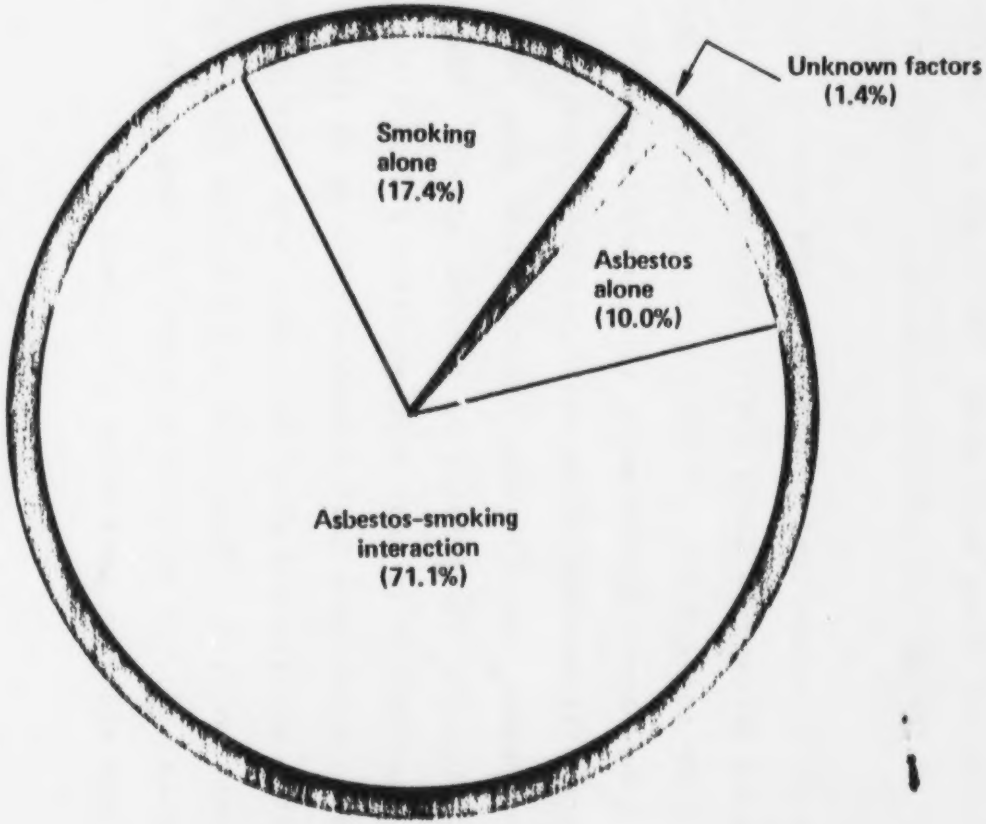


Figure 5. Proportions of Lung Cancer Deaths Attributable to Known and Unknown Factors in a Group of Cigarette-smoking Asbestos Workers

Smoking may also be an important factor in increasing an individual's susceptibility to asbestosis. Although asbestosis occurs in persons who smoke and in those who do not smoke, (e.g, et al. 1979, Hammond et al 1979), Frank (1979) reported that asbestos insulation workers with a history of cigarette smoking had an asbestosis mortality rate 2.9 times higher than that of workers who had never smoked regularly. In addition, several morbidity studies have found that clinical and diagnostic signs of asbestosis in asbestos workers are more prevalent among smokers than nonsmokers (Langlands et al. 1971, Weiss 1971, Weiss and Theodos 1978, Harries et al. 1975, Ayer and Burg 1978, Mitchell et al. 1978, Rossiter and Berry 1978, Berry et al. 1979).

Because none of these morbidity studies included a comparison group of persons who smoked, but who had not been occupationally exposed to asbestos, the "polyvalent" (Becklake 1973) or nonspecific nature of many of the diagnostic signs of asbestosis (i.e., signs that can be produced either by smoking or by exposure to asbestos) could not be taken into account. Consequently, the mortality study (Frank 1979) provides the strongest evidence that smokers are at greater risk of asbestosis than nonsmokers under similar conditions of asbestos exposure.

Data from this mortality study also suggest that there may be a greater risk of pleural mesothelioma among cigarette smokers exposed to asbestos than among nonsmokers similarly exposed (Selikoff 1977a). As shown in Table 12, the rate of pleural mesothelioma mortality among the smokers was more than twice the

Table 12. Mesothelioma Mortality Rates in a Group of Asbestos Insulation Workers, by Smoking History

Smoking history	Mesothelioma deaths/10,000 person–years	
	Pleural	Peritoneal
Never smoked regularly	1.6	7.1
Cigarettes	3.8	7.3
Pipe or cigar	9.7	11.3
Unknown	2.5	3.7

Source: Selikoff (1977a).

rate among the nonsmokers. (There is no evidence that smoking, by itself, can cause pleural mesothelioma.) In contrast, the peritoneal mesothelioma mortality rates were very similar for both cigarette smokers and nonsmokers. The rates in Table 12 were not adjusted for age or duration of asbestos exposure, but they suggest that reevaluation of the conclusion that pleural mesotheliomas "occur with equal frequency among smoking and nonsmoking asbestos workers" (IARC 1977) would be worthwhile. The high mortality rates from both pleural and peritoneal mesothelioma among workers who smoked only pipes or cigars are also worthy of note. Unpublished data supplied to EPA by Selikoff in 1978 indicate that this small group of workers also had a very high asbestosis mortality rate. Although the possible effect of smoking on the risk of pleural mesothelioma should be explored, it is not likely that this effect (if any) will be found to be nearly as large as the effect of smoking on the risk of asbestos-induced lung cancer.

b. Age

The highly active nature of school children and their physical characteristics generate concern that, under similar circumstances, their degree of actual exposure to asbestos may be greater than that of adults (Kane 1976). Because children generally are more active than adults, they have a higher breathing rate. They also inhale relatively more often through the mouth than through the nose. Consequently, more fibers would be inhaled and fewer would be trapped by the nasal hairs and mucosa. Young children are shorter than adults and their mouths and noses are closer to the floor. Therefore, they are likely to inhale higher concentrations of dust that is stirred up from the floor. Children also have a greater remaining life span, during which the chronic effects of asbestos exposure can become manifest.

It has also been suggested that children may be more biologically susceptible than adults to carcinogens, including asbestos (Kotin 1977, Wasserman et al. 1979). Kotin (1976) stated that "...in the induction of cancer, it is the very young that is always the most susceptible." Other observers (Doll 1962, Cole 1977), hold the issue to be far from settled. Kotin (1979) reflected the uncertainty by observing more recently that "special biological susceptibility has not been demonstrated" for children exposed to asbestos.

One epidemiologic study and one experiment with rodents shed some light on this question with regard to pleural mesothelioma. After examining the incidence of this cancer in an epidemiologic study of a group of asbestos textile workers, Peto (1979) stated that "the incidence 30 years after first exposure appears to be much the same irrespective of age at first exposure." The incidence rates were not provided in his report; nevertheless, if the annual incidence is not affected by age at first exposure, then persons exposed earlier in life experience higher lifetime risk. Consistent results were reported in the experiment with rodents by Berry and Wagner (1976), who injected crocidolite into the pleurae of two groups of rats: one at age 2 months and the other at age 10 months. In the group exposed at the earlier age, 40% developed mesothelioma; in the latter group, the incidence was only 19% ($0.005 < p < 0.01$, two-tailed chi square test).

Neither of these studies could evaluate the age-dependent decline in the respiratory clearance of fibers that occurs in humans, at least among smokers (Cohen et al. 1979), and possibly among nonsmokers (Wanner 1977) as well. This decline in clearance capacity might greatly increase the proportion of inhaled fibers that reach the pleura. Therefore, the possibility cannot be ruled out that pleural tissue in young persons may be more susceptible but, because of the relatively unimpaired respiratory clearance in these individuals, less severely exposed than pleural tissue in older persons.

The two studies discussed above apply solely to pleural mesothelioma, which is only one of the hazards of asbestos exposure. The empirical relationship of age at first exposure to the risk of other asbestos-induced diseases remains an unexplored subject.

c. Fiber size and type

A great deal of research and discussion has been devoted to possible variations in risk posed by durable fibers differing in size and chemical composition. Because these factors are not expected to play a major role in the assessment of risks due to asbestos exposure in schools (see part d, Summary and Conclusions, below), they are treated only briefly here.

The primary research relating fiber size to carcinogenic potency applies only to pleural mesothelioma, and it involves the direct injection or implantation of fibers into the pleurae of rats. These studies strongly suggest that fibers of certain sizes are more potent in producing mesothelioma than other-sized fibers of identical or different chemical composition (Stanton and Layard 1978; Stanton 1973; Stanton et al. 1977; Smith et al. 1969; Wagner et al. 1970, 1973 and 1977; Smith and Hubert 1974). As a whole, this research indicates that fibers less than 1.5 microns in diameter and between 5 and 60 microns long, regardless of chemical composition, are likely to be more carcinogenic in the pleura than shorter or wider fibers. The evidence is not sufficient, however, to label fibers with dimensions falling outside this range (especially short, thin fibers) noncarcinogenic.

Fiber size also helps to determine the ability of inhaled fibers to reach the pleura. Because the airways of the lung diminish in size as they branch outward, longer fibers are more likely to become deposited on the ciliated surfaces of the upper airways than shorter fibers (Dement and Harris 1979). This early interception of longer fibers may account for the autopsy finding of a higher percentage of longer fibers in the lung tissue than in the surrounding pleura among persons with asbestos-related disease (Sebastien et al. 1979b). Additionally, longer fibers are less readily cleared from the lung than shorter fibers, especially from the alveoli: the small, saclike pouches that terminate the airways of the lungs (Morgan 1979). Thus, fiber size is an important factor in the transport of inhaled fibers.

Evidence of variation in toxicity according to the chemical composition of asbestos fibers is less firm. There are some indications of slight differences in toxicity among the various types of asbestos (Acheson and Gardner 1979), but these differences may result in part from the different fiber size characteristics of the asbestos types.

There is no evidence that the fiber size distributions to which the epidemiologically studied insulation workers were exposed differed substantially from the distribution of sizes of fibers released from asbestos materials in schools. In addition, all asbestos fiber types found in schools (e.g., chrysotile, amosite, crocidolite) are carcinogenic. Consequently, separate consideration of the health effects of the individual mineralogical types or fiber sizes of asbestos in this assessment is not warranted.

d. Summary and conclusions

Smoking greatly increases the risk of asbestos-induced lung cancer. Although asbestos causes lung cancer in both nonsmokers and smokers, smokers exposed to asbestos have a greater risk of developing this disease than would be expected by adding the separate effects of smoking and asbestos exposure. Smokers also may be at a higher risk of asbestosis than nonsmokers with similar asbestos exposure. Current data on the possible influence of smoking on the risk of asbestos-induced pleural mesothelioma are not persuasive one way or the other. In estimating the risk of lung cancer from exposure to asbestos in schools, smokers and nonsmokers will be considered separately when appropriate data become available from the insulation workers study. Current evidence indicates that most of the increase in lung cancer risk among a group of smokers occupationally exposed to asbestos could have been prevented either by their never having been exposed to asbestos or by their never having smoked.

Although children may be more susceptible to the effects of asbestos exposure than adults, little firm evidence is available to determine the differences in risk. The longer remaining life expectancy of children compared with that of adults is the only factor that can be incorporated into quantitative risk estimates.

Experimental evidence strongly suggests that fibers of certain sizes that reach the pleura, regardless of chemical composition, are more potent in producing mesothelioma than fibers of other sizes. The use of data from a study of asbestos insulation workers for quantitative risk estimates (see Section III, Part D) should avoid any major uncertainties that might otherwise have been presented by this finding. Because there are no data indicating that the fiber types or sizes to which the insulation workers were exposed were substantially different from those present in schools, the types and sizes in both settings will be assumed to be similar.

C. Exposure Assessment

This section assesses the amount of asbestos that inhabitants of schools containing friable asbestos materials are being exposed to by applying current data on airborne asbestos concentrations in various types of buildings to the situation in schools. The results are a quantitative estimate of exposures to the "prevalent" level of asbestos in schools and a qualitative description of exposures to "peak" levels. A description of the methods used to make the quantitative estimates and a discussion of how closely the estimates apply to schools are included.

The prevalent concentration of airborne asbestos fibers is the one present most of the time in areas of activity in buildings. Peak concentrations are those resulting from specific activities such as damage to or repair of asbestos-containing materials, and they generally are high, localized, and of short duration. For our purposes, prevalent levels are those determined by monitoring areas and taking measurements of asbestos concentrations over long periods of time, and peak levels are determined by taking measurements of concentrations resulting from specific activities over short periods of time.

Because area monitoring data are the only consistent data currently available on concentrations of airborne asbestos fibers in buildings and because these data are not likely to include peak concentrations systematically, only exposure to prevalent levels of asbestos in schools can be estimated quantitatively. The area monitoring data do not include peak concentrations systematically for two reasons: (1) peak releases occur sporadically; (2) peak concentrations are limited to very small areas. Only if continuous area monitoring were being conducted at the same time as and very near a specific peak release would the monitoring data reflect peak exposure concentrations. In addition, the possible mechanisms by which asbestos is dispersed in buildings also preclude a quantitative estimate of exposures at peak levels, as explained below.

1. Asbestos Dispersion Mechanisms

The concentration of airborne asbestos fibers is determined by how quickly fibers enter the air and by how quickly they are removed. In buildings containing friable asbestos-containing materials, fibers can be released from these materials and enter the air in several ways (Sebastien et al. 1978, Nicholson et al. 1978a, Sawyer and Spooner 1978). Mechanisms of fiber release such as disturbance of the building materials by air currents will release fibers over a wide area and thus an elevate the prevalent concentration. Other mechanisms of release such as cutting the materials will release a large amount of fibers locally and over a short period of time and, thus, cause peak fiber concentrations (up to several thousand times higher than prevalent concentrations). In addition, fibers that have been removed from the air by settling or by impacting on surfaces (i.e., desks, light fixtures, and floors) can be resuspended in air either diffusely or in the form of peak releases by activities such as dusting, sweeping, maintenance work, etc.

Fibers released during peak episodes eventually become widely dispersed, and are either removed slowly (over periods of hours to days) from the air by settling or impacting on surfaces or are removed when ventilation exchanges indoor and outdoor air. This wide dispersion also elevates the prevalent concentration. Table 13 gives airborne asbestos fiber concentrations that were measured in various buildings (Sawyer and Spooner 1978). The measurements include those of peak concentrations produced by the peak release of fibers directly from asbestos materials (for example, 2f, g, and h in Table 13) and as a result of resuspension (for example, 2c, d, e, i, and j in Table 13).

2. Estimate of Prevalent Exposures

The prevalent exposure levels in schools containing friable asbestos materials were estimated by averaging the asbestos concentrations measured in various buildings. The exposure estimates were based on data from a study by Sebastien et al. (1978) of several buildings in Paris. These data, which are given in Table 14, were not taken in a way which would represent the contribution of peak episodes of exposure. The choice of which specific measurements of asbestos concentration within a building to use in exposure estimates depended on certain assumptions as to what the measurements would represent. Three different assumptions were made to give three different estimates of prevalent exposure (Table 15) that are applicable to all buildings containing accessible friable asbestos materials. A discussion of how these different assumptions apply to exposure in schools and why the data of Sebastien et al. (1978) were used to make these estimates is given below.

Table 13. Optical Microscope Analysis of Airborne Asbestos Fiber Concentrations in Various Buildings

Sampling conditions or situation	Mean counts (f/cm^3)	No. of samples	Standard deviation
1. University dormitory, UCLA. Exposed friable surfaces, 98% amosite. General student activities	0.1	--	0–0.8 (range)
2. Art and Architecture Building, Yale University. Exposed friable ceilings, 20% chrysotile.			
a. Ambient air, City of New Haven	0.00	12	0.00
Fallout			
b. Quiet conditions	0.02	15	0.02
Contact			
c. Cleaning, moving books in stack area	15.5	3	6.7
d. Relamping light fixtures	1.4	2	0.1
e. Removing ceiling section	17.7	3	8.2
f. Installing track light	7.7	6	2.9
g. Installing hanging lights	1.1	5	0.8
h. Installing partition	3.1	4	1.1
Reentrainment			
i. Custodians sweeping, dry	1.6	5	0.7
j. Dusting, dry	4.0	6	1.3
k. Proximal to cleaning (bystander exposure)	0.3	—	0.3
General Activity	0.2	36	0.1
3. Office buildings, Eastern Connecticut. Exposed friable ceilings, 5 - 30% chrysotile.			
Custodial activities, heavy dusting	2.8	8	1.6
4. Private homes, Connecticut.			
Remaining pipe lagging (dry) amosite and chrysotile asbestos	4.1	8	1.8–5.8 (range)
5. Laundry: contaminated clothing, chrysotile	0.4	12	0.1–1.2 (range)
6. Office building, Connecticut. Exposed sprayed. ceiling, 18% chrysotile.			
Routine activity	79[a]	3	40–110 (range)
Under asbestos ceiling	99[a]	2	
Remote from asbestos ceiling	40[a]	1	
7. Urban grammar school, New Haven. Exposed ceiling, 15% chrysotile asbestos.			
Custodial activity: sweeping, vacuuming	643[a]	2	186–1,100 (range)
8. Apartment building, New Jersey; heavy housekeeping. Tremolite and chrysotile	296[a]	1	
9. Office buildings, New York City			
Asbestos in ventilation systems	2.5–200[a]		0–800 (range)
Quiet conditions and rountine activity			

Source: Sawyer and Sponner (1978)

[a] Nanograms/cubic meter. Determined by electron microscope.

Table 14. Measurements of Asbestos Concentrations in Several Paris Buildings Used to Estimate Prevalent Exposure Levels of Asbestos

Building[a]	Sampling sites	Mean conc. (ng/m^3)	Max. conc. (ng/m^3)	Individual samples (ng/m^3)
Ground floor of research building "A"	Parking lots, labs, workshops	215	750	751,518,19,2,0.6,0.
Rooms in research building "A"	Libraries, labs workshops	55	630	630,460,420,225,106, 48,46,37,31,28,15,15 14,13,9,7,6,6,(21 measurements less than 5 ng/m^3)
"B" hangar	Workshops	70	490	492,65,30,24,7,6,5,2,1
"E"	Dining room	29	29	28.8
"H"	Labs, workshops	23	130	134,23,14,12,11,6,5,2,1
"K" railroad station (open walls)	Parking lot (open walls)	16	24	24,12,11
"L"	Mail room	20	34	34,18,17,12
"O"	Laboratory	38	62	62,13

Source: Sebastien et al. 1979.

[a] Designations are those used by the authors.

Table 15. Estimated Prevalent Exposure Levels of Asbestos
(Applicable to all buildings containing exposed friable asbestos materials)

Assumptions used in making estimates	Predicted concn. (ng/m^3)
I. Mean for a building represents the prevalent level	58
II. Maximum for a building represents the prevalent level	270
III. Average of the few highest concentrations for all buildings represents the prevalent level	500

Estimate I is the mean of the mean asbestos concentration in each building (the mean of column 3, Table 14). For each building, this estimate gives equal weight to each measurement of the airborne fiber concentrations but overall Estimate I gives greater weight to individual measurements in buildings where many measurments were taken (e.g. building A). An accurate estimate of population exposure would require that each measurement be weighted according to the number of people exposed at that concentration. Estimate I approximates this by using the mean of the means rather than the mean of all measurements. The measurements listed in Table 14 were made in areas where activities similar to school activities take place, and, therefore, they represent the asbestos concentrations that occur in activity areas in schools. However, they are not accurately weighted according to the distribution of school populations.

Estimate II in Table 15 is the mean of the maximum concentrations of asbestos measured in each building (the mean of column 4, Table 14). The Agency believes this estimate gives more weight to areas of maximum human activity because it is likely that areas where the maximum asbestos concentrations are measured are areas of maximum activity. This hypothesis is supported by data which show that human activity can increase airborne asbestos concentrations by 50 to 200 times (Sebastien et al. 1979a). The major limit to using estimate II is that there is no way to verify that the highest measurements were obtained in the areas of greatest human activity. The overall average exposure to asbestos in buildings containing exposed friable asbestos materials is likely to be between estimates I and II.

Estimate III in Table 15 is the mean of the four highest maxima in column 4 of Table 14 (750, 630, 490, and 130 ng/m^3). Estimate III may account for the future deterioration of friable asbestos materials now in place. Sebastien et al.(1978), describe the sites where the value used in estimate III were obtained as areas where asbestos materials have deteriorated. In the future, friable asbestos materials that are now in place likely will deteriorate through damage or be subjected to maintenance activities such as cutting and drilling. The assumptions in using estimate III to predict future exposure are that deteriorated material is responsible for the few highest measured levels, that all materials eventually will deteriorate, and that when materials do deteriorate, they will cause significantly high prevalent asbestos concentrations. There are insufficient data to document these assumptions.

These three estimates lead to the conclusion that the current average exposure to asbestos in buildings containing accessible friable asbestos materials[6/] is not likely to be less than 58 ng/m^3, it may be as high as 270 ng/m^3, and, in the future, it may become as high as 500 ng/m^3. Of course, these are estimates of exposure to the prevalent concentration. Peak exposures add significantly to the overall exposure of specific groups of people. For example, as shown in Part D, janitors can easily be exposed to an average level of asbestos fibers that is more than twice the prevalent level.

[6/] Materials not enclosed by a solid partition such as a suspended, or false, ceiling.

The estimates are based on data from the Sebastien et al. (1978) study because this study meets the following three criteria: (1) applicability of the data to exposure in schools; (2) consistency, reliability, and accuracy of the measuring and sampling techniques; and (3) adequate data on "control" buildings. The study meets these three criteria because: (1) the areas and materials studied are similar to those in U.S. schools (see discussion below); (2) the measurements were made by transmission electron microscopy (the only technique which is accurate for environmental sampling at low concentrations - see below), the measurements were checked by statistical quality control techniques, and the samples were taken over relatively long time periods (5 days); and (3) comparisons were made with outdoor air and with a significant number of buildings that did not contain asbestos materials. Data on asbestos concentrations in U.S. buildings from a study (Nicholson et al. 1978a) that did not meet these criteria were carefully assessed and used to verify that the results of the Sebastien et al. study are consistent with data for U.S. buildings (Logue 1980).

The specific data selected from the study of Sebastien et al represent the exposure situation in U.S. schools. These data are measurements of asbestos concentrations in buildings with accessible friable asbestos materials. Enclosure of the material (for example, with a suspended ceiling) may greatly reduce exposure, and different enclosures will have different effects. Too little data are available to determine whether the types of enclosures in the buildings sampled by Sebastien et al. are similar to those in U.S. schools. Therefore, exposure estimates are too high for buildings in which enclosed or covered asbestos materials are present.

Friable materials were selected for the estimates because they represent the materials of greatest concern.

Asbestos levels in buildings containing friable asbestos materials are significantly greater than asbestos levels in buildings which do not contain asbestos surface materials. A statistical study (Levy, 1980) showed that there is less than a 5% probability that chance alone caused this difference. Therefore the Agency concludes that the presence of friable asbestos caused the difference. In addition to elevated prevalent exposure in these buildings, peak exposures are likely to be frequent when friable materials are present because these materials are easily damaged.

The selection of friable materials for the estimates does not mean that non-friable materials do not make a significant contribution to both prevalent and peak asbestos exposures. The statistical study cited above, however, shows that there are insufficient data at this time to say whether the observed elevation of asbestos concentration in buildings containing non-friable asbestos materials (see Table 16) is caused by the presence of these materials or due to chance alone. Peak exposures from non-friable asbestos materials can also occur if

Table 16. Comparison of Mean and Maximum Levels of Airborne Asbestos in Buildings Containing Friable and Nonfriable Asbestos Materials[a,c]

Buildings containing friable materials			Building containing nonfriable materials		
Building[b]	Mean concen. airborne asbestos (ng/m^3)	Max. concen. airborne asbestos (ng/m^3)	Building[b]	Mean concen. airborne asbestos (ng/m^3)	Max concen. airborne asbestos (ng/m^3)
A–G	220	750	F	19	40
A-St	55	630	T	21	68
B	70	490	S	0.1	0.1
E	29	29	G	1.7	2.8
H	23	130	I	0.4	2.1
K	16	24	J	3.2	7.1
L	20	34	P	3.2	7.1
O	20	62	Q	0.83	1.3
A–Ct	13	28	R	8.6	12
C	0.17	0.2			
D	3.0	5			

Source: Sebastien et al. (1978)

[a] All asbestos measurements in buildings without asbestos-containing materials were less than 5 ng/m^3.

[b] Building notations are those of Sebastien et al.

[c] Both enclosed and exposed asbestos-containing materials are included in this table.

they are cut or drilled. However, such materials are far less susceptible to damage than are friable materials.

The application of estimates based on various types of French buildings to U.S. schools is possible, because (1) the materials containing asbestos and, as shown by comparing Table 1 and Table 14, the uses of areas in the French buildings are the same as those in U.S. schools; and (2) the French data on asbestos levels in rooms with friable materials are not statistically different from comparable data (Nicholson et al. 1978a) for U.S. buildings (Logue 1980). Asbestos-containing materials in France and the United States are also similar in that the French processes for applying these materials (Sebastien et al. 1978) are similar to the processes used in the United States (cf. Section II of this document). In both cases, "friable" coatings are produced by mixing the asbestos with binders after the material leaves the spray nozzle. In addition, both French and United States data reveal that most of the airborne fibers detected are chrysotile and that the accessible asbestos material is located in similar places.

The use of transmission electron microscopy techniques is necessary for the identification and measurement of asbestos fibers outside of the workplace. The optical microscopy techniques, especially the phase contrast microscopy technique recommended for use in the workplace (HEW 1976), are not suitable for measurement of low airborne asbestos concentrations in buildings because the phase contrast technique cannot distinguish between asbestos and many other fibers and, because the measurement accuracy is limited by the small number of fibers counted (HEW 1976). At low fiber concentrations a larger proportion of the fibers will be non-asbestos fibers and, thus, the ability to distinguish asbestos fibers from other fibers becomes important. For this reason, transmission electron microscopy, supplemented when necessary with electron diffraction to specifically identify asbestos, is the only tool suitable for measuring airborne asbestos concentrations outside of the workplace. 7/

3. Description of Peak Exposures

Significant "peak" releases of asbestos fibers will occur and cause the total exposure of an individual to be higher than the estimated prevalent asbestos concentration. Students, teachers, and school administrators will only occasionally encounter peak exposures. Janitors, custodians, and maintenance workers will encounter them more frequently. Available data are insufficient to estimate the frequency with which either group would encounter peak exposures.

7/ Because both asbestos and non-asbestos fibers are counted, measurements made by phase contrast microscopy are expected to be higher than those made by electron microscopy. This is found in the results of Byron, Hodson and Holms, (1969). They measured fiber concentrations in schools (and other) buildings by the phase contrast microscopy technique and found that 11 of 18 schools with sprayed asbestos had concentrations greater than .005 fibers/cm^3. This corresponds to (using 30 fibers/ng as the conversion) to about 200 ng/m^3 which is much higher than what Sebastien et al. (1978) found by electron microscopy (see Table 14).

Peak exposures occur during episodes of damage to friable asbestos materials, repair or renovations involving the materials, cleaning operations in buildings that contain the materials, or maintenance work performed in spaces that enclose the materials (e.g., in crawl spaces over false ceilings). Peak exposures that occur during damage episodes will affect all occupants, including students, teachers, and school administrators; in general, however, maintenance, janitorial, and custodial personnel will experience peak exposures most frequently. The total exposure of these groups to asbestos may be much greater than their exposure to the prevalent level.

D. Risk Assessment

1. Procedure for Estimating Risks of Premature Death

a. Outline of the Risk Estimation Procedure.

The number of people expected to die prematurely from exposure to asbestos in school buildings can be predicted within limits from available epidemiologic data. The initial step in the risk assessment procedure is to choose the most appropriate epidemiologic study or studies to serve as the basis for making the estimates. The key criteria are that a study contain a quantitative characterization of cumulative asbestos exposure and that the study population exhibit an increase in risk of premature death following asbestos exposure. A statistical model of the relationship between cumulative asbestos exposure and subsequent increases in risk (dose-response model) is then established. The cumulative asbestos exposure of the building occupants is estimated using the prevalent asbestos concentrations (see Part C above), the number of persons likely to be exposed and the probable duration of exposure for those persons. Combining the exposure estimates with the dose-response model allows risk estimates to be expressed as the number of premature deaths expected to occur. In this analysis, such estimates are made for each of three groups of school occupants: students, teachers and administrative staff, and custodians and maintenance workers. Because of the necessary assumptions and uncertainties in quantitative risk assessment, three risk estimates will be presented for each group: the minimum, maximum, and most reasonable predictions of the increases in carcinogenic risk expected to result from asbestos exposure in schools.

Although asbestos exposure in schools likely will also produce signs of asbestosis that are not severe enough to result in death, this risk cannot be estimated quantitatively with currently available data (see Section III, Part B above). In addition, available mortality studies do not reflect the increased incidence of cancer because some cancers (e.g., larynx cancer) frequently are treated with success. These necessary omissions lead to underestimates of the risk of developing nonfatal asbestosis and treatable cancers. This risk assessment is further restricted to a consideration only of exposure to prevalent levels of asbestos in schools. Increased risks resulting from exposure to peak levels have not been included in the overall risk estimate because the frequency of these exposures is unknown.

b. Selection of the Underlying Study.

The epidemiologic study selected to be the basis for making quantitative estimates of the risk of premature death from exposure to asbestos in schools is a large study of asbestos insulation workers reported most recently by Hammond et al. (1979) and Selikoff et al. (1979a). In the original report of mortality among these workers, the men were described as "building trades insulation workers" who were chosen for their "asbestos exposure of limited extent and intensity" (Selikoff et al. 1964).

The data that will be used from this ongoing study concern 12,051 men who were employed in asbestos insulation work for at least 10 years (Hammond et al. 1979, Selikoff et al. 1979a). The results for this group are restricted to the time commencing at the 20th year after each worker's first exposure. each worker's 20th year since first exposure. The diseases caused by asbestos exposure appear after an induction period-- a minimum length of time following initial exposure that must elapse before risk begins to increase. In this study and in others (Peto 1978, Berry et al. 1979, Seidman et al. 1979), the minimum induction period for mortality from asbestos-induced diseases generally has been reported to be 10-20 years. The use of data that cover only the period that starts $\geq$20 years following first exposure allows for the induction of asbestos-induced tumors. The results, therefore, pertain only to the time subsequent to each worker's 20th anniversary of employment, the time at which he was at increased risk of dying from the diseases that are hazards of asbestos exposure.

The mortality experience of the 12,051 workers was observed during the 10-year period from January 1, 1967, to December 31, 1976 (the "follow-up period"). As each of these workers was actively employed on January 1, 1967, and had reached the 20-year point from initial exposure at some time before the end of the follow-up period, each worker was exposed to asbestos for at least 10 years. Workers who had reached the 20-year mark prior to January 1, 1967, were traced throughout the follow-up period (i.e., they entered observation on January 1, 1967). Those who reached the 20-year mark at some time during the follow-up period were followed only from that point on (i.e., they entered observation on the date of the 20th anniversary of employment). The reported increases in risk, therefore, took place ≥ 20 years from initial exposure among 12,051 workers, each of whom previously had been exposed to asbestos for at least 10 years.

In addition to allowing for cancer induction by providing data restricted to the period that starts ≥ 20 years from first exposure, the asbestos insulation workers study has a number of attributes that make it uniquely suitable as a basis for quantitative risk estimation. No other study combines all of these useful attributes:

- The sample of 12,051 workers surviving ≥ 20 years from first exposure is very large, minimizing the probability of chance results.
- Reasonable estimates of the average asbestos concentrations to which insulation workers were exposed are available (see Part c below).
- Each of the diseases identified as hazards of asbestos exposure was investigated and was found to be in excess (see Table 17 below).

- The death certificates were assiduously verified with supplemental information (e.g., autopsy reports, histological specimens) in 86% of the deaths (Selikoff et al. 1979a), leading to a greatly improved detection of frequently misdiagnosed mesotheliomas (Newhouse and Wagner 1969, Selikoff et al. 1979a).
- A highly appropriate control group was used, for which smoking-specific results are available (Hammond 1966).
- The material to which the insulation workers were exposed (commercial asbestos, primarily chrysotile) was very similar and, in some instances, identical to the asbestos present in school buildings.

c. Asbestos Exposure Among the Insulation Workers.

The measure of exposure that will be used in this risk assessment is "cumulative exposure"--the product of the average asbestos concentration (in this study, it is expressed as nanograms per cubic meter of air, ng/m^3) times the number of years of exposure to this concentration. Therefore, cumulative exposure, which is expressed here in units of $ng\text{-}yr/m^3$, incorporates the intensity and duration of exposure into a single measure. This system of measuring exposure has the disadvantage of assuming implicitly that brief, high-intensity exposure is equivalent to extended, low-intensity exposure. For instance, a person exposed to 100 ng/m^3 for 10 years and a person exposed to 1,000 ng/m^3 for 1 year would both be assigned cumulative exposure values of 1,000 $ng\text{-}yr/m^3$. The influence of this assumption on the risk estimates for cancer mortality under the linear nonthreshold dose-response curve will be discussed later.

During the 1940's and 1950's, when the insulation workers in the underlying study received the bulk of the asbestos exposure responsible for risk increases observed during the follow-up period (1967-1976), airborne asbestos concentrations in the work environment probably were higher than the concentrations measured in more recent years. Because reliable monitoring data are not available for the earlier period, approximations must be made on the basis of the recent measurements and in light of changing work practices and conditions. Nicholson (1976) reviewed several monitoring studies and concluded that "the overall time-weighted average exposure of United States asbestos [insulation] workers in the late 1960's was less than 3 f/ml" (3,000,000 f/m^3). This estimate, made under the assumption that insulation workers in the late 1960's worked with asbestos-containing materials only half of the time, agrees closely with the figure of 4,200,000 f/m^3 derived by the National Institute for Occupational Safety and Health (NIOSH 1972) for full time asbestos insulation work. Consequently, 3,000,000 f/m^3 represents the lowest reasonable estimate of the average asbestos concentration to which workers in the underlying study were exposed.

Nicholson (1976) also found that, during the late 1960's, "work practices were virtually identical to those of the past, and ...few controls of significance were in use." Nevertheless, he identified two major changes in the conditions of insulation work over the years. First, workers in the 1940's and 1950's were in contact more often with insulation containing asbestos, as opposed to insulation containing fibrous glass and other materials that recently have become more popular. Second, the asbestos content of insulation materials containing asbestos declined by as much as one-half over the period ranging from the

1940's and 1950's to the late 1960's. These factors helped lead Nicholson (1976) to state that "insulators' average exposures in the United States during the past years could have ranged from 10 to 15 f/ml" (10,000,000-15,000,000 f/m^3). Therefore, 15,000,000 f/m^3 is the highest reasonable estimate of the average exposure level or the asbestos insulation workers.

The most reasonable estimate lies between 3,000,000 and 15,000,000 f/m^3. If it is assumed that insulation workers in the late 1960's handled asbestos one-half of the time, that previous workers handled asbestos three-fourths of the time (a 50% increase), and that older insulation materials containing asbestos had twice the asbestos content of newer asbestos-containing materials, the recent average exposure level (3,000,000 f/m^3) can be multiplied by a factor of 3 to yield an estimate of the earlier concentration. This conversion yields a "most reasonable" estimate of approximately 9,000,000 f/m^3 for the average asbestos concentration to which the workers in the underlying study were exposed.

The units in which the minimum, maximum, and most likely average exposure levels are expressed can be converted from fibers per cubic meter to nanograms per cubic meter. In a study conducted for the Office of Pesticides and Toxic Substances, EPA (Versar 1980), it was concluded that for insulation work, a fiber-to-mass conversion ratio of 30 f/m^3 to 1 ng/m^3 is the best approximation if fibers are counted by light microscopy. This factor is in general agreement with data on fiber size distributions for the asbestos industry as a whole (Dement and

Harris 1979) and for insulation work in particular (Nicholson 1976). It should be remembered that this conversion factor is rough and currently cannot be verified because the nature of the industry has changed. It is, however, the best available estimate.

Conversion of the three estimates of average asbestos exposure for the insulation workers studied by Selikoff's group yields the following estimates:

maximum	500,000 ng/m^3
most reasonable	300,000 ng/m^3
minimum	100,000 ng/m^3

It is important to determine the period of exposure to these concentrations that should be held reponsible for the increases in risk detected during the observation period (1967 through 1976). Asbestos-induced increases in risk do not appear until ≥10 years after exposure (Peto 1978, Seidman et al. 1979), so the attributable exposure period for each worker in this risk assessment ends 10 years prior to the time the worker entered observation.

```
A        B              C                D
|<------>|<----10 yr---->|<-----10 yr----->|
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Under the approach described above, A-B (ending for most workers on December 31, 1956) is the period of attributable exposure. C-D is the follow-up period, during which all exposure is presumed to be "wasted" in the sense that it is not responsible for increases in cancer risk during the same period. B-C is an additional period of time during which exposure is considered to be "wasted." If the minimum induction period for death from asbestos-induced neoplasms were exactly 10 years, the greatest degree of underestimation of exposure would result from the fact that exposure during year B+1 ended, not 10 years, but 18 years before year D-1 began. The choice, however, of a length of 10 years for the additional "wasted" exposure period (B-C) is a conservative one. The minimum induction period for mesothelioma, for instance, appears to be closer to 20 years than to 10 years (Selikoff et al. 1979a). For lung cancer, Peto's (1978) data show the minimum induction period to be about 15 years. If the minimum induction period for these diseases were >20 years, no relevant exposure would be ignored by choosing 10 years for the length of period B-C.

Thus, although a certain degree of exposure relevant to increased risk during the follow-up period (C-D) is likely to be ignored under this approach, 10 years for B-C is considered a reasonable length in order to optimize three goals: (1) to make use of the published mortality data from the insulation workers study; (2) to avoid attributing "wasted" exposure to increased risk during the follow-up period; and (3) to avoid labeling exposure "wasted" that actually contributed to increased risk during the follow-up period. The attributable exposure period for each of the 12,051 workers was his period of employment ending 10 years before he entered observation. The total number of years of relevant exposure for the group divided by 12,051 yields the average exposure period. [Note: We are requesting this total from the researchers. For the time being, a figure of 20 years will be used as the mean attributable exposure period in the calculations. When the actual value becomes available, it will replace the 20-year figure.]

It was estimated above that the average exposure level for the insulation workers was between 100,000 and 500,000 ng/m^3, with the most reasonable estimate being 300,000 ng/m^3. Over a 20-year average exposure period, these figures yield the following estimates of cumulative exposure:

maximum	1.0×10^7 ng-yr/m^3
most reasonable	6.0×10^6 ng-yr/m^3
minimum	2.0×10^6 ng-yr/m^3

The three values are estimates of the average cumulative asbestos exposure of the 12,051 workers at the time 10 years before observation began. Many continued to be exposed, but exposure beyond that point is not thought to have contributed to the observed increases in risk.

d. Increased Risk Among the Asbestos Insulation Workers.

Following the 20-year induction period, the researchers compared the observed number of deaths from specific causes among the 12,051 workers to the number expected on the basis of mortality rates in an appropriate comparison group.[8/] The greater number of observed than expected deaths indicates increased risk. The results for cancer deaths are shown in Table 17. The use of 95% confidence intervals for the observed number

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[8/] The data for the control or comparison group were obtained from a large study sponsored by the American Cancer Society (Hammond 1966) of the age-, calendar year-, and smoking-specific experience of white males with at most a high school education and a history of occupational exposure to dust, fumes, vapors and gases, excluding farmers.

Table 17. Mortality Data Taken from the Study of 12,051 Asbestos Insulation Workers and Used To Make Quantitative Estimates of Risk from Asbestos Exposure in Schools

Cause of death	Expected deaths (E_i)[a]	Observed deaths (O_i)	95% Confidence limits[b]		Statistical significance level[c]
			Lower	Upper	
Cancer, all asbestos-related sites	145.8	692	641.4	745.6	<0.001
Lung	81.7	397	358.9	438.1	<0.001
Pleura	0	61	46.6	78.4	<0.001
Peritoneum	0	109	89.5	131.5	<0.001
Larynx, buccal cavity, pharynx	7.5	21	13.0	32.1	<0.001
Esophagus	5.1	17	9.9	27.2	<0.001
Kidney	8.5	15	8.4	24.7	0.027
Colon-rectum	30.5	54	40.6	70.5	<0.001
Stomach	12.5	18	10.7	28.4	0.084

Source: Hammond et al. (1979)

[a] Number of observed deaths based on death certificate information only, except for pleural and peritoneal mesothelioma. Supplemental information was used for these two cancers. This procedure was recommended by Hammond et al. (1979).

[b] Method of Bailar and Ederer (1964), assuming a Poisson distribution of observed deaths. Values from Documenta Geigy (1970), some by linear interpolation.

[c] Method of Bailar and Ederer (1964), assuming a Poisson distribution of observed deaths. One-tailed test, values from Molina (1942).

of deaths is a way of accounting for the role of chance variation in the results. In comparisons with the expected number of deaths, the minimum risk estimate is provided by the lower 95% confidence limit for observed deaths, the maximum estimate by the upper 95% confidence limit for observed deaths, and the most likely risk estimate by the actual number of deaths observed.

The measure of increased risk most useful for predicting premature mortality from asbestos exposure in schools is the difference between the observed (0_i) and expected (E_i) numbers of deaths from the cancers related to asbestos exposure divided by the total number of deaths expected from all causes ($E_T = 1,148.0$) (Hammond et al. 1979). This measure is the fraction of all expected deaths that were "in excess" because of the asbestos exposure. It is called "lifetime risk" (LR) by the EPA Carcinogen Assessment Group, Office of Research and Development, EPA, and is defined as follows: $LR= (0i-Ei)/E_T$. If the study were carried out until all were deceased (actually, only 16% of the 12,051 workers died during the observation period), (0_i-E_i) would equal the total number of "excess," or premature deaths.

In using lifetime risk, as defined above, as the measure of increased cancer risk in this assessment, certain assumptions must be made. First, it must be assumed that the estimate of lifetime risk when only 16% of the workers have died will be the same when all 12,051 have died. Second, it must be assumed that this estimate of lifetime risk among persons exposed as adults will be indicative of the lifetime experience of exposed school children, who have a greater remaining period of expected life during which the effects of asbestos exposure can become manifest. The use of the lifetime risk measure, therefore, does not allow the greater remaining life expectancy of children to be taken into account and for this reason may underestimate risk.

The mortality rates for each of the cancer hazards of asbestos exposure were increased among the insulation workers (Table 17). The data for the separate causes of death can be combined in order to estimate overall lifetime risk for all asbestos-induced cancers:

maximum = (745.6 - 145.8)/1,148.0 = 0.522

most reasonable = (692 - 145.8)/1,148.0 = 0.476

minimum = (641.4 - 145.8)/1,148.0 = 0.432

The results give a most reasonable estimate that the overall mortality rate was increased by 48% above the expected value by asbestos-induced deaths from cancer. (Additional premature deaths from asbestosis are not included here.) The above lifetime risk estimates will be used to predict the risks of mortality from asbestos exposure in schools.

e. Asbestos Exposure in Schools

In Section III, Part C, three prevalent asbestos concentrations were estimated for school buildings. Estimates I (58 ng/m^3) and II (270 ng/m^3) were developed to reflect current concentrations and Estimate III (500 ng/m^3) to reflect concentrations in the future, as the building materials deteriorate. The risk assessment concerns exposures over the next 30 years. Consequently, Estimates I and III will be used as the minimum and maximum future concentrations, respectively. Estimate II, although developed as a maximum estimate of the current concentration, will be used as the most reasonable estimate of future concentrations.

Three groups of school building occupants are considered: students; teachers and administrative staff; and custodians and maintenance workers. It was estimated in Section II that approximately 3,000,000 students, 222,000 teachers and administrative staff, and 23,000 custodians and maintenance workers are occupying schools that contain friable asbestos materials (See Section II above). These numbers of exposed school occupants at risk of death from asbestos-induced cancers need to be adjusted to reflect the number expected to die before a minimum period of time from first exposure has elapsed. Adopting the same 20-year minimum induction period as in the insulation workers study and assuming that the average student is first exposed at age 12 and the average adult school occupant at age 30, national life tables (NCHS 1978) can be used to estimate that 2.2% of exposed students and 5.9% of exposed adults will die before 20 years have elapsed from first exposure. The estimated number of school occupants at risk, then is 3,000,000 - 2.2% = 2,934,000 persons exposed while attending school, 222,000 - 5.9% = 208,900 teachers and administrative staff, and 23,000 - 5.9% = 21,600 custodians and maintenance workers. The average remaining service time for the buildings is approximately 30 years (See Part II above). Therefore, the average cumulative exposure for the adult occupants is equal to the prevalent asbestos concentration times 30 work years.[9/] For students, it is the prevalent concentration times 15 work years. These estimates are shown in Table 18.

The calculations were made under the assumption that each of the current school occupants will be exposed for the entire 30-year period that the buildings will remain in service. Although this assumption is not technically correct (as students graduate and adults leave their positions, others will replace them), as long as the exposed populations continue to average 3,000,000, 222,000, and 23,000, respectively, the risk estimates will not be affected. This is because of the nature of the cumulative exposure measure (1,000 ng-yr/m^3 resulting from either 10 years at 100 ng/m^3 or 1 year at 1,000 ng/m^3) and the linear nonthreshold dose-response model (cumulative exposure of 1,000 persons to 1,000 ng-yr/m^3 yielding the same number of premature deaths as cumulative exposure of 100 persons to 10,000 ng-yr/m^3).

f. Selection of the Extrapolation Method

Once the most suitable epidemiologic study of asbestos workers has been chosen for the risk assessment, a method must be selected for using the results of the study to predict the

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[9/] A work year is assumed to be made up of 50 weeks at 5 days a week and 8 hours a day. A school year is assumed to be made up of 33 weeks at 5 days a week and 6 hours a day. Therefore, 2 school years equal one work-year:

$$\frac{33 \text{ weeks}}{50 \text{ weeks}} \times \frac{5 \text{ days}}{5 \text{ days}} \times \frac{6 \text{ hours}}{8 \text{ hours}} = 0.5.$$

Table 18. Estimates of Cumulative Asbestos Exposure in Schools[a]

Population group	Average number exposed	Cumulative exposure levels ($ng\text{-}yr/m^3$) for: Minimum estimate of risk	Most reasonable estimate of risk	Maximum estimate of risk
Students	2,934,00	870	4,050	7,500
Teachers, administrative staff	208,900	1,740	8,100	15,000
Custodians, maintenance workers	21,600	1,740	8,100	15,000

[a] Assuming 30 work years of exposure per adult and 15 work years of exposure per student. See discussion on page 92.

increase in carcinogenic risk that will result from asbestos exposure in schools. The prediction is made by extrapolating[10/] from the exposure levels experienced by the asbestos insulation workers to the lower levels of asbestos to which school occupants are exposed. A dose-response curve is developed to describe the relationship between cumulative asbestos exposure and subsequent increases in cancer risk. From this curve, predictions of increased risk can be derived that correspond to cumulative exposure levels lower than those for which epidemiologic data are available. Because the empirical relationship of dose to response at these exposure levels is unknown, criteria must be established for selecting the most appropriate dose-response curve.

Two general types of evidence can be used to show that a dose-response curve or model is unsuitable: (1) knowledge of the biological processes that influence the degree to which inhaled asbestos increases carcinogenic risk, and (2) the dose-response data available from epidemiologic studies or experiments with laboratory animals. The first type of information, often called "pharmacokinetics," includes a carcinogen's "absorption, distribuiton, reactions with cellular components, and elimination," as well as its interaction with physiologic

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[10/] If, as in most risk assessments, it is assumed that the dose-response curve passes throught the point corresponding to zero exposure and zero increase in risk, the prediction is technically an interpolation. Nevertheless, the conventional term, extrapolation, will be used here.

mechanisms of activation and detoxification (Gehring et al. 1977). This kind of information, if available, can lead to inferences about the shape of the dose-response curve at low doses, including the possibility of a threshold dose below which the risk of cancer would not be increased (Cornfield et al. 1977). EPA is unaware of information about the pharmacokinetics of asbestos that would enable such inferences to be drawn.

The second type of evidence, dose-response data from epidemiologic and toxicologic studies, is available for asbestos-induced carcinogenicity. Table 19 shows the results of statistically "fitting" several dose-response models that have been developed for chemicas carcinogens to data from two studies: an epidemiologic study of asbestos workers (Henderson and Enterline 1979, see also Figure 1-B) and an experiment with rats (Wagner et al. 1974). By the usual and widely accepted criterion that a p-value greater than 0.05 or 0.10 indicates an adequate fit (Remington and Schork 1970), none of the models can be dismissed on the basis of these studies.

Current scientific evidence alone cannot be used to select the most appropriate dose-response curve for this extrapolation because none of the curves in Table 19 can be ruled out on the basis of pharmacokinetics or available dose-response data. Of these curves, however, linear nonthreshold regression (see, e.g., Figure 1) usually provides the highest predictions of increased risk and there is no strong scientific reason to prefer any of

Table 19. Dose-response Curves Applied to Two Studies of Asbestos Exposure and Carcinogenic Response

		Goodness-of-fit p-value[d]	
Dose-response curve	Reference	Epidemiologic study[a,e]	Experiment with rats[b]
One-hit	Brown (1976)	0.58	0.08
Multi-hit	Van Ryzin and Rai (1980)	0.42	0.13
Multi-stage (1 stage)	Crump (1980)	0.58	0.11
Multi-stage (5 stages)	Crump (1980)	0.53[c]	0.09[c]
Linear regression	Neter and Wasserman (1974)	0.87	0.10

[a] Henderson and Enterline (1979)

[b] Wagner et al. (1974)

[c] The results of a Monte Carlo simulation

[d] The "p" values calculated from the chi-square (χ^2) statistic are based on the difference between the observed (O_i) and the expected (E_i) counts in the ith dose group. The degrees of freedom equal number of dose levels-1-(number of parameters estimated).

$$\chi^2 = \sum (O_i - E_i)^2 / E_i$$

[e] In the epidemiologic study, each measure of response concerns a group of people with a unique age distribution; hence, the "background" mortality rates will differ among the groups. To fit models with this type of data, it is necessary to adjust the observed response to what they would be if there were a common "background" rate. The risk attributable to the carcinogen is calculated from Abbott's equation:

$$P = (P_{dose} - P_{control}) / (1 - P_{control})$$

The adjustment to common "background" is done by recalculating the observed response, P_{dose}, as P'_{dose}. Where $P'_{control}$ represents the common "background" rate:

$$P'_{dose} = P(1 - P'_{control}) + P'_{control}$$

the dose-response curves that yield lower estimates of increased risk. Consequently, the Agency has chosen to take the most prudent course with respect to public health by using a linear nonthreshold dose-response curve to extrapolate from the asbestos exposure levels experienced by the insulation workers to the lower levels of asbestos to which school occupants are exposed.

2. Risk Estimates for School Building Occupants.

Figures from Tables 17 and 18 and the preceding discussion are summarized in Table 20. They are arranged in the way that yields minimum, most reasonable and maximum estimates of increased risk for school building occupants.

When the linear nonthreshold model is used, calculation of predicted risk levels is fairly simple. For instance, as shown in Table 21, the minimum lifetime cancer risk for school children would be:

$$(0.432 \times 870 \text{ ng-yr/m}^3)/(1.0 \times 10^7 \text{ ng-yr/m}^3) = 3.8 \times 10^{-5}.$$

Multiplying this figure by the number of students yields the minimum estimate of premature deaths:

$$(3.8 \times 10^{-5}) \times (2,934,000) = 111.$$

Table 20. Summary of Cumulative Exposure and Lifetime Risk Estimates To Be Used in Quantitative Risk Assessment of Asbestos Exposure in Schools

	Estimates leading to:		
Population group	Minimum risk estimate	Most reasonable risk estimate	Maximum risk estimate
		Cumulative exposure (ng-yr/m^3):	
Insulation workers	1.0×10^7	6.0×10^6	2.0×10^6
Students	870	4,050	7,500
Adult schools occupants	1,740	8,100	15,000
		Lifetime cancer risk (%)	
Insulation workers	43.2	47.6	52.2

Table 21. Quantitative Risk Estimates of Mortality from Exposure to Asbestos in Schools

Population group	Lifetime risk			Premature deaths		
	Minimum	Most reasonable	Maximum	Minimum	Most reasonable	Maximum
2,934,000 students	3.8×10^{-5}	3.2×10^{-4}	2.0×10^{-3}	111	960	5,868
208,900 teachers, administrative staff	7.5×10^{-5}	6.4×10^{-4}	3.9×10^{-3}	16	142	814
21,600 custodians, maintenance workers	7.5×10^{-5}	6.4×10^{-4}	3.9×10^{-3}	2	15	84
			Totals	129	1,117	6,766

The results of this and similar calculations are shown in Table 21. A total of approximately 100 to 7,000 premature deaths are anticipated to occur as a result of exposure to prevalent concentrations of asbestos in schools containing friable asbestos materials over the next 30 years. The most reasonable estimate is approximately 1,000 premature deaths. About 90% of these premature deaths are expected to occur among persons exposed as school children. The remaining 10% include teachers, custodians, and other adult occupants of the buildings. The most reasonable estimates represent extrapolations of approximately four orders of magnitude from the exposure levels experienced by the insulation workers.

The risk estimates in Table 21 are subject to further refinement. For instance, the influence on risk of the greater remaining life expectancy of children compared with that of adults has not yet been incorporated into the assessment. In addition, when supplemental information requested from the investigators who conducted the insulation workers study is supplied, it will alter these estimates to some degree. The information requested is:

- the total number of person-years of exposure accumulated by the 12,051 workers up to the time 10 years before they entered observation;
- the number of person-years of observation and the number of observed and expected deaths from lung cancer, asbestosis, and pleural mesothelioma by smoking history;
- the number of observed and expected deaths due to cancer of the colon separate from those due to cancer of the rectum.

This additional information, with the possible exception of the smoking-specific data for lung cancer, is not expected to have a major effect on the overall results of the risk assessement. At present, an assumption is implicit that the distribution of smoking habits among exposed school occupants will be the same as among the insulation workers.

It is important to emphasize that the risk estimates in Table 21 concern only a portion of the total adverse impact on health expected to result from asbestos exposure in schools. The less-than-fatal effects of asbestos exposure on lung function and the number of cases of certain types of cancer that may be treated successfully (e.g., larynx cancer) are not included in this quantitative risk assessment. The substantial but unquantifiable risks resulting from peak exposures also are absent from the assessment.

IV. IDENTIFICATION OF FRIABLE ASBESTOS-CONTAINING MATERIALS IN SCHOOLS

A. Introduction

In order to control the release or resuspension of asbestos in schools or other types of buildings, it is necessary to determine whether asbestos is, in fact, present in the bulk building materials. This determination can be made by examining building records and by analyzing samples of the materials. Records will not establish conclusively that asbestos is not present in a building, as they may be incomplete or there may have been a substitution (e.g., of asbestos for nonasbestos fibers) of the components in the material that was applied. The magnitude of the risks involved makes it necessary to take additional, more extensive steps such as sampling/analysis to ensure the accurate identification of friable asbestos-containing materials.

To locate friable materials, it is necessary to visually inspect the steel support beams, columns, ceilings, and walls of all areas of the school. Asbestos-containing materials also may have been applied to hidden areas, such as those above a suspended ceiling, and they must be checked. Inspectors should direct particular attention to boiler rooms and other equipment areas, in view of the frequent use of asbestos as insulating material.

Procedures for inspecting buildings and taking samples of friable materials and guidelines for establishing the presence of asbestos in these materials are described in two recent EPA publications (EPA 1979b and EPA 1980, respectively).

B. Sampling

A sample of friable material can be obtained by penetrating the depth of the material with a small canister or jar or by dislodging the material with a knife. An amount equal to 2 tablespoons is sufficient for analysis. Proper sampling requires that each sample container be tightly sealed, wiped clean with a damp cloth, and labeled. The label should be recorded by the sampler (EPA 1979b).

Samples must be taken in a manner that will provide a representative indication of the composition of the material. The amount of asbestos in the friable asbestos-containing materials on a building surface may vary. If the material is homogeneous in appearance and was applied at one time, the amount may not vary greatly over one surface area. From three to seven samples may be needed, however, to establish whether asbestos is present and, if so, the approximate percentage that is present. EPA recommends that 3 samples be taken for homogeneous surfaces that are up to 1,000 ft^2, 5 samples for surfaces that are between 1,000 and 5,000 ft^2, and 7 samples for surfaces that are >5,000 ft^2 (EPA 1980).

A random selection of sampling sites is necessary to eliminate the bias that may result from taking samples from convenient locations. Representative sampling can be achieved by extracting material from different places within a sampling area (close to walls, at joints, etc.). A more involved method for the random selection of sample sites (EPA 1980) involves the use of a random number table and a diagram of the area to be sampled.

Friable material is disturbed during the sampling process, and asbestos fibers, if present, may be released. Release of and consequent human exposure to asbestos can be minimized by taking samples when the area is not in use and limiting the number of persons present, lightly spraying water on the area to be sampled to discourage dust formation, holding the sample container away from the face, and wet cleaning the area if any pieces are dislodged and fall to the floor.

C. Analysis

Three analytical methods can be used to identify asbestos fibers in bulk materials. The first, polarized light microscopy (PLM), uses the different refractive indices, birefringence, and other optical crystallographic properties of asbestos minerals to distinguish them from nonasbestos ones. PLM also characterizes and identifies other fibers such as glass fibers and cellulose. The second method, x-ray diffraction (XRD), uses the unique diffraction pattern produced when x-rays strike any crystalline material to identify specific asbestos minerals. The third, electron microscopy (EM), uses electron diffraction or energy-dispersive x-ray analysis to identify asbestos fibers by examining the structure of individual fibers.

EPA's Guidance Manual on Asbestos Analytical Programs (EPA 1980) recommends PLM as the method of choice for determining asbestos in suspect material and XRD as a backup technique to confirm the PLM analysis. Although electron microscopy can be used, it is not recommended, because only very small quantities of sample can be analyzed at one time and the analysis of multiple samples is prohibitively expensive.

The EPA Environmental Monitoring Systems Laboratory, Research Triangle Park, North Carolina, has prepared and currently is field testing interim PLM and XRD analytical protocols to be followed in identifying asbestiform minerals in bulk samples. The protocols clarify and refine the guidance originally offered in Appendix H of the Guidance Manual. They have been circulated to laboratories currently participating in the Technical Assistance Program. An Asbestos Particle Atlas with color PLM photomicrographs has been developed by McCrone Research Institute. The Atlas is available from Ann Arbor Press.

EPA has identified and complied a list of laboratories that analyze bulk samples for asbestos using PLM. This list is based in part on the laboratories' successful participation in a proficiency analytical testing program. A report on this testing program will be available in September, 1980. Copies of the list can be obtained from EPA by calling the following toll-free number: 800-344-8571, extension 6892.

V. CONTROL OF ASBESTOS IN SCHOOLS

This section presents information on the steps that can be taken to control exposure to asbestos in school buildings once friable asbestos-containing materials have been identified.

EPA has published guidance materials on the corrective actions that can be taken in schools and other buildings if asbestos-containing materials are found to be damaged or deteriorating. Long-term solutions to the release of asbestos fibers from these materials are removal, encapsulation, or enclosure. Removal eliminates the source of contamination, enclosure (with a barrier such as a suspended or false ceiling) reduces the likelihood that incidental contact with the asbestos-containing material will occur, and encapsulation (with an effective sealant) reduces the likelihood that fibers will be released into the building environment.

Exposure to asbestos in buildings also can be controlled to some extent by a number of other actions, most of which are aimed at reducing physical contact with asbestos-containing surfaces. These actions simply interrupt the process by which asbestos fibers enter building air. Asbestos fibers enter a school environment from friable asbestos-containing materials as a consequence of:

(1) disturbance of the material during maintenance or renovation operations, implementation of the long-term corrective actions described above, and vandalism;

(2) fallout encouraged by normal activity in the building; and

(3) resuspension of settled fibers caused by normal activity or custodial dusting or cleaning.

Usually, asbestos enters the air as a result of physical contact with asbestos-containing material. Contact can cause significant amounts of fibers to be released to the air, resulting in airborne concentrations that frequently exceed industrial standards (Sawyer 1977).

Loosely compacted friable materials are more likely to release fibers than tightly bound materials. When a friable material was brushed by hand to simulate mild damage, fiber counts as high as 3.8 f/cm^3 were measured as far away as 10 feet from the site of the damage (Nicholson et.al. 1978a). In contrast, counts of 0.2 f/cm^3 were noted when a cementitious material was brushed (Table 17 in Nicholson et.al.).

Repair, renovation, or maintenance of buildings may bring about the highest airborne concentrations of airborne fibers, because these activities disturb asbestos-containing materials directly. Sanding or cutting asbestos-containing solid materials during construction or repair produces the greatest release of fibers. Incidental contact that occurs when other maintenance chores (e.g., installing a lighting unit) are performed can lead to significant release. In addition, damage to the material from vandalism, maintenance work, or, simply, deterioration can increase the rate of fiber release by fallout (Sawyer 1977). In schools, there is the additional opportunity for damage of friable materials by students. Whether it is the result of normal school activity (such as throwing a ball around a gymnasium) or acts of vandalism, damage caused by students can be significant.

Small amounts of asbestos can fall spontaneously from ceilings or walls, building up the airborne fiber concentration over time. Low harmonics and vibrations caused by machinery and other sources also can increase the release of fibers. Once a wall or ceiling is damaged, it can shed fibers without significant further disturbance. These fibers can accumulate around a room and be continually resuspended any time there is movement of the air. Accumulation of asbestos fibers caused by fallout can be significant.

Finally, the resuspension of fibers that have been released can continue to cause asbestos exposure. Cleaning or other maintenance work or the movement of people through an area can cause settled fibers to be resuspended. Suspended ceilings can hide the accumulation of fibers until maintenance work causes the suspended ceiling to be disturbed; this could result in the release of a large amount of fibers to the air (Sawyer 1977). Custodial services such as sweeping and dusting also can elevate fiber levels by disturbing material that has collected on floors and other surfaces. Asbestos fibers tend to stay suspended in the air for a long time; for smaller fibers, this time may be on the order of days. When they do settle out, the fibers can easily be resuspended. They do not diffuse as a gas does; rather, they tend to be confined to a given area.

Exposure to asbestos can be controlled to some extent by reducing the physical contact of individuals with friable asbestos-containing materials. Sawyer reported on the beneficial effects of wet cleaning, wet handling during maintenance, and barrier systems in inhibiting the movement of fibers in a building. The simple rearrangement of schedules so that direct work on asbestos-containing material will occur when the building is not in use and provision of workers with respirators also can reduce inhalation of asbestos. Regular wet cleaning of building surfaces can remove accumulated fallout, thus reducing the resuspension of asbestos. Sawyer reported that wet cleaning reduced fiber concentrations due to custodial activity from 4.0 f/cm^3 (before control) to 0.3 f/cm^3. Wet cleaning is particularly effective in reducing the exposure of the person doing the cleaning.

General exposures throughout the building also might be reduced somewhat as a result of wet cleaning, although no studies have been done to show the effectiveness of regular wet cleaning per se on the building environment. Unless care is taken in disposing of any fibers collected during either wet or dry cleaning, fibers will remain available to be resuspended in building air.

Sawyer reported that during removal operations, wetting bulk asbestos-containing materials with water containing wetting agents reduced mean fiber counts to 8.1 f/cm^3, compared with the mean count of 82.2 f/cm^3 that was calculated for dry conditions. [Nicholson et al. (1978a) reported fiber counts of up to only 1.78 f/cm^3 during wet removal of asbestos-containing materials in a New Jersey school.] Sawyer also demonstrated that fiber levels dropped more quickly when wet methods were used.

The migration of fibers to non-work areas can be inhibited by barriers. When removing asbestos in a New Jersey school, Nicholson et al. isolated the work area with plastic barriers.

Fiber counts outside the work area ranged from 0.01 to 0.03 f/cm^3, but counts within the removal area ranged from 0.02 (during wetting) to 1.78 f/cm^3.

Vacuum cleaners equipped with high-efficiency particulate absolute (HEPA) filters can collect asbestos dust. Sawyer showed that, whereas dry dusting of shelves and books in a library raised fiber counts to 4.02 f/cm^3, use of HEPA filters raised counts to only 0.4 f/cm^3. Wet wiping the shelves produced a count of 0.2 f/cm^3. Household and normal industrial vacuums without HEPA filters cannot collect asbestos fibers.

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